To My Three Masters in Medicine

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WHO BY THEIR TEACHINGS, WRITINGS AND PERSONAL FRIENDSHIP HAVE BEEN MY INSPIRATION

PREFACE TO THE SEVENTH EDITION

It is gratifying that the successive printings of the sixth edition of this book were so quickly exhausted, and that a seventh edition is now required. This rapid growth denotes usefulness

To be useful, a textbook must be alive. To keep it living, it must embrace facts that are time-honored and proved to be true, as well as the new facts that are evolved by progress. Consequently, the author of such a text must periodically discard the decadent and add the modern

This seventh edition is my sixth opportunity for exercising an author's privilege of correcting and adding to the information in the preceding editions. The chapters on physical diagnosis remain unaltered. In the other chapters, however, much new material has been included, and it is felt that this will materially enhance the value of the book. Among these new discussions, the following subjects should be mentioned: infection and immunity, thermometry and methods of converting the various thermometric units, etiology of fever, Loeffler's syndrome, cysts of the lungs, sarcoidosis, virus pneumonia, rheumatic pneumonia, mycotic pneumonitis, the peritoneum and its anatomy and the various types of peritonitis, ascites and its types and differential diagnosis, the Rh factor in human blood and manner of transmission and role in pregnancy, the Hr antigens, transfusion reactions, puberty, menstruation, and the menopause, puberty and the male climacteric, Peyronie's disease, Reiter's syndrome, Cruveilhier-Baumgarten's syndrome, Lasègue's sign, Pitfield's sign, and Rivalta's test for globulin.

There are new tables also, and among them are the following subjects: the incubation periods of febrile diseases, the thermometric equivalents of the three types of thermometers, the kinds of specimens to be collected for laboratory examinations, the diseases transmitted by arthropods and their causative agents In addition, several new illustrations appear.

It had long been known that the title, "Medical Diagnosis and Symptomatology," used in previous issues of this book, was an inadequate expression of its content. Much of the book is concerned with physical diagnosis as well, and this dual approach has at last demanded the more meaningful title, "Medical and Physical Diagnosis"

I should like to express my appreciation to my wife for her usual care in reviewing the manuscript, to Mr. Wendell H. Grenman, of the F. A. Davis Company, for his care and suggestions, and to my publishers for their patience and encouragement.

SAMUEL A. LOEWENBERG

PREFACE TO THE FIRST EDITION

Despite the present trend of medicine towards extreme specialization, the author has ventured to compile a text-book of general information upon medical diagnosis from the standpoint of the rapidly disappearing "general practitioner." His reason for bringing forth a book of this type is his belief that no one can become a real specialist until he has practiced general medicine long enough to enable him to view human ills from the standpoint of "*the person* affected by an illness" rather than "*the illness* affecting a person." It is not the author's intention to advocate a retrogression in medicine or a reversion to an older type of "jack of all trades and master of none," but rather to encourage more masters whose judgment has become mature by reason of the experience gained both from general practice and from a chosen specialty. Because of the interrelation of all parts and organs in the human body no one part or organ alone can be treated successfully unless proper consideration is given to the organism as a whole. Therefore the specialist, no matter how expert he may be in his own field, must nevertheless have a knowledge of general medicine.

Oliver Wendell Holmes likens the brain to an attic where old furniture, bric-a-brac and other odds and ends are stored away; and, in order to make room for more things, some of those previously stored must be discarded. Likewise in order to acquire new knowledge some of the old must be removed or forgotten. If we accept the simile, then let us hope that the candidate for specialism has first acquired adequate knowledge of the various phases of medicine and thereby learned to discriminate wisely as to what to discard in order to make way for the fuller knowledge of the particular branch of medicine which holds his special interest. Experience gained in the practice of general medicine will mature his judgment sufficiently to appreciate the value of his discards so that he does not throw away material more valuable than he acquires

This book aims to cover the field of diagnostics in internal medicine It gives instructions on the various methods of examining the patient, descriptions of normal findings, enumeration of pathologic conditions with the normal and pathologic physical signs, and, whenever possible, the reasons for such signs. The signs and interpretations are discussed from the viewpoints of the medical student, the general practitioner and the specialist. The respiratory and cardiac systems are discussed fully and minutely; to the digestive system, the nervous system and urology, adequate space is devoted, while to the skin, nose, ears, eyes, bones and joints,

radiography, the blood, the ductless glands, etc., less space is given, only so much being allotted as is deemed necessary for the purpose of a general examination. The chapter on laboratory interpretations is limited, in the main, to the interpretation of laboratory analyses reported by the pathologist, chemist, serologist or clinical laboratory specialist, while only the simplest technical methods are described. The chapter on life insurance examination, the examination of industrial workers, periodic health examinations and the detection of malingering deals chiefly in generalities, as the specific methods of examination are amply described in other chapters The illustrations are of three types: (1) actual photographs of methods of examination and of patients suffering with the particular disease described in the text, (2) drawings calculated to emphasize the descriptions of certain conditions, and (3) photographs of pathologic specimens to aid the memorizing of the respective clinical descriptions.

The author hereby acknowledges his indebtedness to the authors of various text-books and of articles in the current medical literature bearing upon the subject matter of this book, from which sources he has quoted freely, credit being given in the text wherever these quotations and opinions appear. He is especially grateful to Milton K. Meyers, M.D., for the preparation of the Chapter on Neurology; to Leon Solis-Cohen, M.D, for the preparation of the Chapter on Roentgenology; to Max Trumper, Ph D, for the revision of the work in Hematology; to Solomon Solis-Cohen, M.D., for his many suggestions while the manuscript was in preparation; to the Pathological Department of the Philadelphia General Hospital for the majority of the photographs appearing in this book; to Mr H N Gosner, photographer at the Philadelphia General Hospital; to my publishers, the F. A Davis Company, and to others who by their work, advice and friendship have made this volume possible

SAMUEL A. LOEWENBERG

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CHAPTER I

Preliminary Considerations and History Taking

I. Introduction

The practice of medicine is founded upon two essentials, diagnosis and treatment. Of these, diagnosis is the first and most fundamental; for, depending upon *it, the particular type of treatment necessary* to overcome the disease is instituted. Since diagnosis is the cornerstone of medical science upon which the entire superstructure of its modern practice has been built, to master it is, or should be, the aim of everyone who undertakes the art of healing. It is true that the chief end in view is to overcome disease by treatment, but this must be based upon a recognition of the seat and the nature of the abnormal processes to be remedied. That knowledge can be reached only by a careful and thorough examination.

A complete diagnosis has been divided by S. Solis Cohen into four phases, or, as he terms them, partial diagnoses (1) *The symptomatic or clinical diagnosis,* based on the characteristic features of a given clinical type of case, (2) the lesional or pathologic diagnosis, which concerns itself with the site of the original lesion; (3) the physiologic or functional diagnosis, which has to do with the manner in which observable disturbances of functions are produced; and (4) the etiologic or causal diagnosis, dealing with the specific cause or causes of the disease in question.

To satisfy completely all these postulates, one would have to master every intricacy of the diagnostic art. Therefore, it is not very often that anyone is ready to say, or to feel assured, that he has arrived at a complete diagnosis. The best that one can do in many instances is to approximate this ideal as closely as possible, and to embrace every opportunity for study, practice and investigation

*The basis of diagnosis is symptoma*tology and physical examination; but it is also true that there are other means of ascertaining the presence and cause of disease, and that in certain conditions our final decision must be based upon supplementary methods such as the roentgen rays and other laboratory aids. Yet, useful as these are, it is still to symptomatology and physical examination that we are obliged to look for our chief source of information. The availability of chemical and instrumental aids to diagnosis has tended to make physicians undervalue the importance of skill in physical examination, and to mislead students into the belief that time spent in acquiring *such skill is today of small importance.* The fallacy of such reasoning will soon become apparent to the physician whose work leads him away from the big centers of population or from the well-equipped city hospitals. The man who has put his faith in x-ray machines, calorimeters, electrocardiographs, etc., and failed to perfect himself in the art of physical examination, will find that his labor has been largely misdirected. As the great clinician Harvey Cushing wisely stated:

"We have instruments of precision in increasing numbers with which we and our hospital assistants at untold expense make tests and take observations, the vast majority of which are but sup-

plementary to, and as nothing compared with, the careful study of the patient by a keen observer using his eyes and ears and fingers and a few simple aids. The practice of medicine is an art and can never approach being a science even though it may adopt and use for its purposes certain instruments originally designed in the process of scientific research."

Many mistaken diagnoses result from insufficient or faulty history, evaluation of symptoms and physical examination. It is incumbent upon the physician to examine the patient's entire body, and not to rest content with investigating only that part of it to which the patient himself has directed notice; nor even to confine his attention to a particular area where he may have detected some irregularity that conforms to his or the patient's preconceived idea of the cause of the trouble. Many an otherwise keen observer and excellent diagnostician is possessed of some obsession, particularly so if he specializes in one of the branches of medicine, so that he approaches every patient with a preconceived diagnosis and attempts to so interpret the patient's symptoms as to make them fit that diagnosis. There are few morbid states that do not present at least one symptom, which, to a mind filled with a particular clinical picture, can indicate the disease which holds his special interest. No matter how thorough the examination, the interpretation will be colored by this preconceived idea.

The practitioner who has thoroughly mastered the art of eliciting an adequate history and of conducting a physical examination must then bring to his work not only a skilled hand and a trained eye, but a free and open mind. Then only may he hope to interpret correctly what he feels and sees, and sum up the evidences of his senses with unbiased judgment. Only such an attitude can approximate the ideal of a complete diagnosis.

II. Evaluation of History or Anamnesis

Among the requisites for a correct diagnosis is the eliciting of a careful "history." The history should include all the information obtainable concerning the development of the patient's illness up to the time the physician first sees him, as well as a description of the symptoms which are in evidence at the time of examination; also a history of previous illness, and of familial predisposition.

Questions are to be framed so that the patient finds it a simple matter to give accurate answers. It is, however, best to refrain from asking "leading questions," thus avoiding the filling of the patient's mind with any obsession. At the same time the physician should carefully side-step any possibility of falling into the same pitfall. To learn the type of questions to be asked and the manner of approach requires time and experience which may be gained through consultation and interviews with patients seen in daily practice.

The physician has to learn to discount many of the statements made to him in regard to past illnesses, and has to look with suspicion upon the nomenclature which the average nonmedical person attaches to previous indispositions from which he may have suffered. This is especially true of such terms as "rheumatism" or "nervous disorders." Patients are often also apt either to exaggerate or to underestimate pre-

vious ailments, or may deny a previous infection or venereal disease The experienced diagnostician listens patiently to all that is told him, and believes as much of it as circumstances warrant and the physical examination corroborates and often has to surmise what is left untold Thus, it will be seen that skillful history taking requires the finesse of a diplomat and the tact of a father-confessor, to say nothing of a very good knowledge of men and of medicine. Such things cannot be done by rote, and no textbook can teach them.

The patient's history will disclose the type of disease from which he suffers, whether acute, subacute or chronic, and will indicate the kind of studies required in order to diagnose his ailment. A proper diagnosis can be made only after evaluating the history and the symptoms presented by the patient together with a thorough physical examination and such laboratory and special examinations as may be suggested by the history, the symptomatology and the physical signs.

In order to follow the course of an illness, and to note its progress and the value of the treatment, and to be able to formulate a prognosis, it is often necessary to review daily the progress of the disease and to note the condition of the patient, the development of new symptoms and signs and to obtain from the attendant a history of all that had occurred since the physician's previous visit. In many instances it is necessary to evaluate daily or oftener the physical signs of the affected parts and of the vital organs and to repeat certain of the laboratory examinations; and, when necessary, to have new tests made. It is a good plan for the student of medicine to develop, as early as possible, keen powers of observation so that he may become acquainted with the physiognomy of disease Certain diseases so stamp themselves upon the individual as to endow him with definite characteristics. The importance of seeing what one is looking at cannot be overestimated.

III. History Taking

Identity: The name, address, occupation, sex, age, nationality and marital condition of the patient are to be recorded.

Chief Complaint: The patient is interrogated as to his ailment and the chief complaints and his answer is written down in his own words

Family History: This includes the medical history of father, mother, brothers, sisters, uncles, aunts, cousins—if living, health of each; if dead, cause of death and age at which it occurred. Inquiry should also be made about any diseases that may run in the family, especially with reference to tuberculosis, diabetes, gout, epilepsy, cancer, hypertension, apoplexy, mental disorders, cardiovascular disease, digestive disturbances and endocrinopathies

Personal History: This includes the history of the patient from birth to the present time. Inquiry is made as to the following:

Diseases of childhood and complications, if any;
Diseases of adolescence and adulthood, especially venereal diseases;
√Operations or serious injuries,
Habits—tobacco, alcohol, drugs, tea, and coffee;
Masturbation during youth;
√Past occupations;
Place of birth—rural, suburban, or urban, and size of community;
Countries in which patient has resided;
Social condition, and, if married, health of spouse, number of children and their health; if any children are dead, cause of death;
Any miscarriages.

Then inquiry is made specifically regarding the patient's past general health with reference to the following systems:

Gastrointestinal System: This is investigated as to the appetite, the extent to which food is chewed, "indigestion" and symptoms, such as nausea, vomiting, belching, regurgitation, dysphagia, "heartburn," abdominal pain and its radiation and relationship to ingestion of food; jaundice, hematemesis, as well as regards the condition of bowels, type of stool, color of stool and whether bright red or black at any time; and hemorrhoids. The weight is investigated as to the best, the average and the present weight of the patient.

Respiratory System: Here it is important to note susceptibility to colds, sore throat, tonsillitis or quinsy. Inquiry is also made as to: Hoarseness, cough and expectoration; type and time of cough and whether coughing spell is ever followed by vomiting; hemoptysis; odor and amount of expectoration; night sweats; shortness of breath; pain during respiration.

Cardiovascular System: Inquiry should be made as to shortness of breath on exertion; the amount of exertion necessary to bring on dyspnea; the occurrence of orthopnea; precordial pain, its radiation and the relationship between precordial pain and exertion; edema of ankles, choking sensation in neck, syncope or vertigo and any cardiac palpitation; also whether or not the patient is conscious of missed beats or paroxysmal tachycardia, of throbbing sensation in the neck, and of the occurrence at any time of hemoptysis or hematemesis.

Urinary System: Attention should be paid to the presence or absence of headaches, edema of eyelids, blurred vision, frequency of urination—day and night, burning on urination, incontinence, difficulty in starting or stopping stream, distortion of stream, the occurrence of hematuria, the color, quantity and odor of the urine and, in women, whether coughing, laughing or sneezing is accompanied by spurts of urine.

Nervous System: Inquiry should be made as to the vision of each eye, the hearing capacity, the presence of otorrhea, tinnitus aurium, and vertigo, palsies or tremors, and of areas of anesthesia, hyperesthesia, paresthesia or myesthesia. The emotional state is to be investigated, noting the presence of depressions, expansions, indifferences, hallucinations, illusions, delusions, fears and phobias and the state of memory. The patient's station, gait, and his ability to walk in the dark or with eyes closed are also to be noted. If headache is present, the location, intensity and causes are to be investigated. Inquiry is also made as to sleep, *i. e.*, whether soundly, fitfully or restlessly, etc., and as to the occurrence of dreams and their nature.

Gynecological System: Inquiry should be made as to: The menses—when established, regularity, duration, pain, changes if any, and last appearance; vaginal discharge—amount, duration, color, consistency; pregnancies—number full term, abortions, character of labors, convalescence, subsequent health; menopause—gradual or sudden and any complications; coitus, if painful and methods employed to avoid conception.

Genitourinary System: Inquiry is to be made as to: Venereal infection, such as gonorrhea and chancre; time of

infection, nature of treatment received, when cured, presence or absence of complications and sequelae; also the history as to masturbation, sexual life, potency and perversions.

History of Present Illness: Special attention is paid to the history of the present illness as to date of beginning, cause (patient's view), prodromes, specific and general complaints, treatment previous to present examination, etc.

A complete history is usually taken at the first visit of the patient. At times, however, with a nervous patient or with one who is too sick or reticent to disclose the past or the family history, this may be obtained at subsequent visits. Patients suffering from chronic ailments or those *requiring* a complete examination, such as a periodic health examination, or for any other reason may be subjected to a detailed history as is indicated in the following form:

DETAILED HISTORY FORM

1. Name — Country of Birth
2. Address — White — *Colored*
3. Age — Single — Married — Widowed — Divorced
4. What is your present occupation
5. Have you changed your work frequently — Why
6. What are the conditions of your work
   Regular — Dangerous — Dark — Smelly — Seated — Hours per day
   Satisfactory — Fatiguing — Light — Noisy — Standing — Days per week
   Monotonous — Indoors — Out — Dusty — Crowded — Walking
7. Are your earnings *sufficient* to support yourself and dependents comfortably
8. What are your home conditions
   In a *family* — Congenial — Quiet — Room and bed to yourself
   Alone — Depressing — Irritating — Time to yourself
9. What are your sleeping conditions
   Hours in bed — Windows open — Restful — Disturbed
10. How often do you eat
   Regularly — Where — Between meals — Time of meals
11. Are you a moderate or hearty eater, taking one or more helpings at a meal of
   Meat (including fish and eggs) — Pie, Cake or Pastry — Salads
   Baked beans — Sweets or Sugar — Bread
   Green vegetables (spinach, cabbage, etc.) — Fruits — Butter
   Potatoes (*rice*, macaroni or cereal)
12. How much do you drink daily of
   Milk — Tea — Soft drinks
   Water — Coffee — Alcoholic drinks
13. How frequently do you use candy — How much tobacco
14. Do you have a movement of the bowels daily — With the use of drugs
15. What exercise do you take in addition to your work
16. What are your social, political, club or trade associations
17. What are your pleasures — Recreations — Hobbies
18. Are you subject to worries — Moods — Periods of alternating gloom and cheerfulness

19 Have you ever been ill with any of the following, or any other severe illness and at what ages

Tuberculosis	Syphilis or Gonorrhea	Typhoid Fever	Convulsive Seizures
Malaria	Scarlet Fever	Tonsillitis (Sore Throat)	Nervous Breakdown
Rheumatism	Diphtheria	Frequent Colds	Migraine or Neuralgia

20 Have you been protected against smallpox typhoid diphtheria or other diseases by vaccination and when

21 Have you had any accidents, broken bones or surgical operations

22 How often do you consult your dentist When last

23 Are your parents, brothers and sisters living
If not, what were the causes of death and at what ages

24 Have either of your parents or any brother or sister or any of your playmates or associates had consumption cancer insanity epilepsy gout diabetes

25 Do you consider yourself in good health If not, what is your complaint

For Women

26 Are your monthly periods regular Prolonged Excessive

27 Have they interfered with your occupation In what way

28 Have pregnancies and confinements been free from accident

IV. Age and Sex in Reference to Disease

Not only is it necessary to differentiate broadly between infancy, early childhood and adolescence, but it is important to consider the approximate age of the patient, because certain diseases are more prevalent at certain periods of life, and also because premature senility or, occasionally, prolonged immaturity may be an expression of pathologic conditions. It has been said that a man is as old as his arteries and that a woman is as old as she looks, but in actual practice the examiner should carefully compare the appearance of the patient with the age given. Rapid aging may cause a man of 35 to appear 60, while a man who is really 60 may, because of inherited vigor and proper hygienic living, be as powerful physically as a man of 35. Premature senility may be due to privation, dissipation, physical or mental strain, or inherited structural defects Immaturity may be caused by endocrine disturbance.

The following is a table (alphabetically arranged) of some of the commoner diseases, listed under the period of life in which they are most likely to occur, though any of the diseases mentioned in this table and many more not here mentioned may occur at any period of life.

Diseases of Infancy and Childhood: Acute anterior poliomyelitis Affection of lymph glands (tuberculosis) Chorea. Congenital syphilis. Convulsions. Cretinism. Endocarditis. Exanthemata (measles, scarlet fever, smallpox, etc.). Foreign bodies in respiratory and deglutitory passages. Hydrocephalus Infantile paralysis. Infantile palsies (especially birth palsies). Helminthiasis Hypertrophic pyloric stenosis. Inflammation of the respiratory system. Intussusception. Infantile forms of muscular atrophy and muscular dystrophy. Laryngeal diphtheria. Laryngismus stridulous Lobular pneumonia. Meningitis. Mumps. Otitis media. Progressive muscular atrophy. Pseudohypertrophic paralysis. Pyelitis Virus infections.

Diseases Common to Adolescence: Acne. Addison's disease. Anemia. Acute appendicitis. Catalepsy. Chlorosis. Dementia precox. Epidemic encephalitis—acute. Epilepsy. Gastric ulcer. Goiter—

in its various forms. Graves' disease. Hysteria—various forms Juvenile forms of muscular atrophy and dystrophy. Juvenile paresis Mitral and Aortic disease. Multiple sclerosis Paresthesia—various forms. Pneumonia. Rheumatic fever—acute. Sarcoma Tonsillitis—acute (also Quinsy). Tuberculosis. Typhoid fever and other acute infections.

Diseases Common to Middle Age: Angina pectoris. Aneurysm. Apoplexy. Arteriosclerosis. Asthma. Bright's disease, chronic. Bulbar paralysis. Cancer. Emphysema and chronic bronchitis. Gallstones and gallbladder disease. Goiter. Gout Hypertension Hypochondriasis Involutional melancholia. Leukemia Melancholia Myocarditis. Paralysis agitans. Pernicious anemia. Pregnancy and the disorders incidental to it. Presenile dementia. Progressive spinal muscular atrophy. Pseudoleukemia. Sciatica. Syphilis. Tuberculosis—chronic. Valvular heart disease.

Diseases Common to Old Age: Aortic disease Apoplexy. Bronchitis, chronic. Bronchopneumonia Cancer. Cerebral disease Emphysema. Myocardial disease Prostatic disease. Senile dementia

V. Evaluation of Symptoms

Symptoms as applied to disease are subjective evidences or manifestations of pathologic processes. They are abnormal functional phenomena felt by the patient but may not always be perceived by the examiner. Disease may be diagnosed by one of three methods or by all three methods, namely· Symptomatology; physical signs, and laboratory investigation Primarily the patient consults the physician because of the occurrence of some abnormal phenomena. The physician gathers all the data concerning these abnormal sensations by the anamnesis; he then investigates these symptoms by a properly conducted physical examination, and if further study is necessary the aid of laboratory methods is sought

Symptoms may be divided into *general* and *pathognomonic.* General symptoms are those that may occur in many abnormal conditions and by themselves are not diagnostic of any particular disease Pathognomonic symptoms are those that always occur in a disease; their presence indicates a particular or specific disease Among the commoner symptoms for which patients seek relief are *fever, pain, abnormal sensations, digestive disturbances, weakness, dyspnea, cough, nervousness, etc.*

The history of the present illness as well as the morbid manifestations occurring during the course of an ailment are largely a recitation and observation of symptoms. Many symptoms are pathognomonic of certain diseases, while others have no specific significance and may be found in many diseases At times the presence of several symptoms in an illness, though each symptom when occurring alone is nonspecific, may constitute a pathognomonic symptom complex or a syndrome.

A symptom may be defined as a subjective sign felt by the patient and not always perceived by others. A sign is an objective manifestation Often symptoms and signs are dependent upon each other or are so intimately combined that it is difficult to separate them. The symptoms present in various diseases may either be sufficient to make a diagnosis or they may indicate the kind of examinations and studies to be carried out so as to arrive at a diagnosis Symptoms may be general, local or specific

General symptoms are pain, fever, chills, sweats, etc.

Local symptoms may be general symptoms localized in specific areas, such as pain in the head, in the joints, etc., or symptoms occurring in disease of certain systems, such as the digestive system, the cardiovascular system, etc.

Specific or pathognomonic symptoms are those occurring as specific characteristics of a disease as, for example, night blindness in retinitis pigmentosa and slow adaptability to light in vitamin A deficiency.

For symptoms in detail, see the following chapters

VI. Evaluation of Physical Signs

A complete physical examination should be made at the first visit unless the patient's condition is such that the strain of undergoing it would be too severe, as is often the case after a hemorrhage or in extreme exhaustion and in extreme nervousness. Under such circumstances, as much of an examination is made as is consistent with the patient's condition and the necessity of establishing a tentative diagnosis.

Every student should familiarize himself with the methods of physical examination, and practice them systematically It has been well said: "More errors arise from want of system than from want of knowledge." One should always adopt a carefully conceived plan of physical examination and adhere to it religiously.

The physical examination begins with general observations as soon as the physician and patient meet. If the patient is in bed the posture should be noted, also the expression of the face as to whether it gives evidence of pain or other emotion. A considerate bedside manner and a kindly approach reassures the patient and inspires confidence. Especially is this important with a patient who is acutely ill or one who is suffering from a psychoneurotic ailment. Because of the hypersensitivity of such bed patients it is often advisable to obtain the history from an attendant and not in the presence of the patient. The patient may then be asked a few relevant questions before the physical examination is begun. Occasionally he may be voluble and insist upon relating every symptom, real and imaginary. Under such circumstances the physician must listen patiently and at the same time observe the patient's behavior, mannerisms, color, etc. After having obtained a history, the temperature is tested, the pulse is counted and the physical examination is carried out methodically and without seeming haste.

In ambulatory patients also, much of their reticence and self-consciousness may be dispelled by a friendly attitude and tact on the part of the physician. A few cheerful remarks will usually put the patient at ease, and while the history is inquired into the physician has an opportunity to observe the patient's behavior as to restlessness, diffidence or overboldness, the manner of dress, cleanliness, etc. After the history has been obtained, the patient should remove as much of his clothing as the physical examination may require. For further details see Chapter VI, pp. 107 to 123

VII. Evaluation of Laboratory and Special Examinations

After a careful history has been taken and a thorough physical examination has been made of the patient, it often becomes necessary, for the sake of arriving at a correct diagnosis, to employ

certain instruments of precision and to have the patient's secretions, excretions and various tissues examined by laboratory means.

Urinalysis and blood examinations should be made as a general routine in practically all cases. Other laboratory examinations, such as bacteriological, serological, radiographic, etc., are employed according to the indications as obtained from the history and physical examination of any given case. For further details, see Laboratory Chapters XXXIII to XXXVII, inc., p 967 to end.

VIII. Indications for Laboratory Examinations

Diseases of the Kidneys: ***Urinalysis:*** Urinalysis includes observation of color, odor, and specific gravity; test for alkalinity or acidity; tests for albumin (coagulation test, ring test); microscopic examination of urinary sediment.

Renal Function Tests: Urine concentration tests (Fishberg, Mosenthal, and Lashmet and Newburgh); urinary excretion tests (urea clearance and phenolsulfonphthalein); examination of the blood for urea nitrogen, nonprotein nitrogen, creatinine; other tests when required, *i e.,* blood, amino acids, chlorides, and carbon dioxide; examination of urine for acetone bodies; and an Addis count for urinary sediment.

Diseases of the Liver: The following tests are employed to determine the functional capacity of the liver: icterus index; Van den Bergh; bromsulphthalein; hippuric acid; galactose tolerance; levulose tolerance; Takata-Ara test; cephalin precipitation; colloidal gold; iso-iodokon, cholesterol; vitamin K determination; examination of the bile and total blood protein.

Diseases of the Stomach: Gastric analysis and x-ray studies

Diabetes Mellitus: Examination of urine for sugar, acetone, and diacetic acid; examination of blood (fasting) for glucose, glucose tolerance.

Diseases of the Meninges: Cerebrospinal fluid examination for general appearance; culture; Wassermann; colloidal gold; chlorides; glucose, cell count.

Fever: Complete blood count; urinalysis and various tests, depending upon the clinical findings, *i e,* blood culture, skin tests, agglutination tests, microscopic examination of stained or of unstained specimens of blood.

SECTION 2

Symptomatology

CHAPTER II

Temperature Alterations, Sweats, and Chills

I. Infection and Immunity

Infection: Most of the febrile diseases are caused by pathogenic bacteria that are transmitted directly or indirectly by carriers to nonimmune (susceptible) persons The portals of entry may be the respiratory tract, the digestive system, the genital tract, the blood stream, the mucous membrane, or the skin unbroken or broken by various wounds, abrasions, scratches, punctures, incisions, bites, or stings In some diseases the portal of entry or the mode of infection is not definite known During the prodromal stage of a disease, the micro-organisms, having gained entrance to the body, propagate and multiply in large numbers, and invade the blood stream and other tissues of the body.

Many of the organisms have a predilection for various structures, i.e. the typhoid bacilli for Peyer's patches, the diphtheria bacilli for the mucous membranes of the pharynx and the larynx, the pneumococci for the lungs, the meningococci for the brain, and the various fungi for the skin and mucous membrane The reaction of the body to the various organisms may be severe or mild, local or general.

Severity of the Reaction (Infection): This depends upon the following: (1) the virulence of the micro-organisms, i.e. the greater the virulence the more severe the infection; (2) the number of the invading organisms, i e. the greater the number the more severe the infection; and (3) the susceptibility of the host, i.e. his reaction to specific infective organisms. The severity of the reaction also depends upon relative, acquired, or natural immunity. A person who has a marked degree of immunity towards certain organisms or their toxins is less likely to become infected by them than the one who lacks that degree of immunity, and when infected the reaction is comparatively mild

Spread of Infection: This depends largely upon general immunity and local tissue resistance. The infection may be local or general.

1. *Local Infection:* This may occur when a tissue has become partially devitalized in a person whose general immunity is sufficiently strong to prevent a systemic spread of the infection. When the organisms are avirulent, or the general immunity is strong, the infection will remain localized (focal infection). Should the immunity be overcome by the virulence of the local invasion, the weakening of the natural barriers will allow the infection to enter the blood stream and cause widespread or generalized infection, such as septicemia, pyemia, or bacteriemia. This may occur in streptococcic, staphylococcic, and certain other microbic infections. When general immunity is moderately strong and the local tissue resistance is weak, the infection will remain confined, but will not become systemic. In the absence of immunity to a specific infection, local invasion by the specific micro-organism will cause a rapidly spreading systemic infection.

2. *General Infections:* These comprise specific fevers and intoxicants. The portal of entrance may be a local-

temperature is considered to be 98.6° F (37° C) This may vary somewhat at different periods of the day in the same person, and it may be at a constant higher or lower level in different persons throughout the day.

The temperature is measured with the clinical thermometer. It may be inserted in the mouth far back underneath the tongue; in the rectum about 3 cm (1¼ inches), or until it slips past the internal sphincter; in the axillae or in the groin (here care must be taken to hold the thermometer tightly in place by the arm or thigh respectively) The length of time indicated for keeping the particular instrument in place should be doubled.

Normal Temperatures:

Mouth 98.6° F. (37° C).
Rectum: 99.2° F (37.3° C)
Axillae 98° F (36.5° C)

Normal Ranges of Temperature:

Mouth: 97° F (36° C) to 99.2° F. (37° C).
Rectum 97.5° F (36.3° C) to 100° F (37.7° C).
Axillae: 96.5° F. (35.5° C) to 97.5° F (36.3° C).

This temperature range is maintained regardless of the temperature of the surrounding atmosphere unless it becomes either extremely hot or extremely cold. Thus, when a normal, naked man is exposed to temperatures ranging from 40° F to 105° F., his internal temperature does not vary perceptibly. If exposed to a temperature below 40° F., he will develop chills which help to increase the production of his bodily heat, and when exposed to the higher degree of the range, he will sweat, thus increasing his heat dissipation As long as the balance between heat production and heat dissipation is maintained, the body temperature remains constant. Women have a more efficient heat regulating mechanism than men; therefore they can withstand extremes in temperature with less discomfort than men (Hardy and DuBois).

Thermometry. ***Types of Clinical Thermometers:*** There are three types of clinical thermometers in use in various countries: the Fahrenheit, the Centigrade, and the Reaumur.

1. *The "Fahrenheit" (F.)*: This has the freezing point of water at 32° and the boiling point of water at 212°. This type of thermometer (oral or rectal) is common in the United States of America, Canada, England, and several other countries

2. *The "Centigrade" (C.)*: This has the freezing point of water at 0, and the boiling point of water at 100°. This type is in common clinical use in many of the European, Asiatic, and African countries, and in most of the scientific works throughout the world

3 *The "Reaumur" (R)*: This has the freezing point of water at 0, and the boiling point at 80°. At present this type is used in but few countries.

Conversion of Various Thermometric Units: To convert Centigrade to Fahrenheit, multiply by $9/5$ and add 32 Example. C. $100 \times 9/5 = 900/5 = 180 + 32 = 212°$ F.

To convert Fahrenheit to Centigrade, subtract 32 and multiply by $5/9$. Example: F. $212 - 32 = 180 \times 5/9 = 900/9 = 100°$ C

To convert Reaumur to Fahrenheit, multiply by $9/4$ and add 32 Example: R $80 \times 9/4 = 720/4 = 180 + 32 = 212°$ F

To convert Fahrenheit to Reaumur, subtract 32 and multiply by $4/9$. Example: F. $212 - 32 = 180 \times 4/9 = 720/9 = 80°$ R.

To convert Reaumur to Centigrade,

multiply by 5/4. Example· R. 80 × 5/4 = 400/4 = 100° C.

To convert Centigrade to Reaumur, multiply by 4/5. Example: C. 100 × 4/5 = 400/5 = 80° R.

The conversion of the degrees of one scale to those of another may be expressed in the following formulas:

F. = 9/5 (C. + 32) = 9/4 R. + 32.
C = 5/9 (F. — 32) = 5/4 R.
R. = 4/9 (F. — 32) = 4/5 C.

Approximate Thermometric Equivalents

Fahrenheit	*Centigrade*	*Reaumur*
96 8	36 0	28 8
97 0	36 5	29 2
97 5	36 6	
98 0	36 7	
98 6	37 0	29 8
99 0	37 3	
99 5	37 5	30 0
100 0	37 8	
100 4	38 0	30 4
100 8	38 2	
101 0	38 4	
101 3	38 5	30 8
101 6	38 7	
102 0	38 9	
102 2	39 0	31 2
103 0	39 5	31 6
103 5	39 7	
104 0	40 0	32 0
104 5	40 3	
104 9	40 5	32 4
105 0	40 6	
105 5	40 8	
105 8	41.0	32 8
106 0	41 2	
106 5	41 4	33 12
107 6	42 0	33 6

Classification of Temperature

(*a*) Subfebrile temperatures vary from 99 5° to 100 4° F. (37 4° to 38° C).

(*b*) Slight fever, 100 5° to 101.1° F (38° to 38 4° C.).

(*c*) Moderate fever, 100 2° to 102 2° F. in the A. M to 103° F., P. M. (38 5° to 39° C. in the A M to 39 5° C, P. M.).

(*d*) High fever, 102.3° F. in the A M. to above 104° F. in the P M. (39° C. in the A. M. to above 40° C. in the P. M.).

(*e*) Hyperpyrexia, when the temperature rises above 105 8° F. (41° C.).

(*f*) Subnormal temperature, below 97° F. (36 1° C).

Hyperpyrexia: This may occur in sunstroke, the temperature rising to 107° or even to 110° F.; occasional brief periods of hyperpyrexia may occur during the course of acute infections, and not infrequently a sudden rise of temperature to the hyperpyrexia level may occur following certain cerebral injuries, in some of the acute infectious diseases, or it may occur just before death

Stages of Fever: The course of an attack may be divided into four stages: (1) The prodromal stage; (2) the onset or invasion; (3) the fastigium or stadium, and (4) the decline or defervescence.

1 ***The Prodromal Stage:*** The length of time varies in different infections. Prodromal symptoms are chiefly noted in diseases of gradual onset; often they appear several days to several hours before the development of fever. In some of the fevers, prodromal symptoms are absent The usual prodromal symptoms are lassitude, vague aches and pains, headache, digestive disturbances, irritability of the nervous system, and mild subfebrile temperature. In some of the exanthemata a prodromal rash precedes the characteristic eruption, *i. e*, in smallpox, a punctate rash on the inner aspects of the thighs; in measles, Koplik's spots over the buccal mucous membrane; in scarlet fever, very red throat; and in chickenpox, a generalized erythematous scarlatiniform rash.

The Period of Incubation· Preceding the onset of fever, after exposure to infection, there is a latent period known as the *incubation period* This represents the time elapsing between the entrance of the infective agent into the body and the appearance of the major clinical manifestations of the disease. The period varies in different diseases, and often in the same disease in different persons.

Approximate Incubation Periods of Various Febrile Diseases (Alphabetically Arranged)

Diseases	*Incubation Period*
Amebiasis (intestinal)	4 to 90 days
Anterior poliomyelitis acute (infantile paralysis)	Average 10 days, though it may be shorter or very much longer
Anthrax	1 to 7 days
Asiatic cholera	Few hours to 6 days
Brucellosis (undulant fever)	1 to 4 weeks, average 2 weeks
Chickenpox (varicella)	14 to 21 days
Common cold	A few hours to a few days
Dengue	4 to 10 days
Diphtheria	2 to 5 days
Dysentery (amebic)	4 to 90 days
Dysentery (bacillary)	1 to 8 days
Encephalitis lethargica (epidemic encephalitis)	4 to 15 days
Epidemic meningitis (spotted fever, cerebrospinal fever, meningococcic meningitis)	2 to 10 days, usually 7
Epidemic pleurodynia	2 to 4 days
Epidemic streptococcus (septic sore throat)	1 to 3 days
Erysipelas	1 to 3 days
Foot-mouth disease (aphthous fever)	2 to 14 days
German measles (rubella)	10 to 22 days
Glanders (Farcy de Boeuf)	A few hours to 3 weeks, generally about 4 days
Gonorrhea	3 to 15 days
Haemophilus influenzae pneumonia	2 to 5 days
Haverhill fever	Uncertain, possibly 10 to 15 days
Hookworm disease	Uncertain, 2 weeks to 2 months or longer
Influenza	1 to 3 days
Kala azar	Uncertain (2 weeks to 4 months)
Leprosy	Uncertain
Lymphogranuloma inguinale (lymphopathia venerea)	10 to 50 days
Malaria tertian (Plasmodium vivax)	Prodromal symptoms on the 6th day
Malaria tertian (Plasmodium ovale)	About 14th day 14 to 17 days

Approximate Incubation Periods of Various Febrile Diseases (Alphabetically Arranged)—Continued

Diseases	Incubation Period
Malaria quartan (Plasmodium falciparum)	10 to 12 days
Measles (morbilli)	11 to 14 days
Mononucleosis infection (glandular fever)	7 to 10 days
Mumps (epidemic parotitis)	17 to 21 days
Oroya fever (Carrión's disease, bartonelliasis, verruga peruana)	14 to 21 days
Pappataci fever (Phlebotomus fever)	2½ to 6 days rarely 9 days
Paratyphoid fever	8 to 12 days
Pertussis (whooping cough)	7 to 14 days
Plague (bubonic)	2 to 10 days
Plague (pneumonic)	1 to 3 days
Pneumonia (pneumococcal)	1 to 2 days
Pneumonia (virus, presumably)	1 to 21 days
Poliomyelitis	7 to 14 days
Psittacosis	8 to 14 days
Puerperal sepsis	1 to 3 days
Rabies (hydrophobia)	10 days to 2 or more years, average 50 to 60 days
Rat bite fever	5 to 14 days up to 6 weeks, average 13 days
Relapsing fever (tick fever)	2 to 15 days average 7 days
Rocky Mountain spotted fever	4 to 8 days
Salmonella suipestifer infection	6 to 48 hours
Scarlet fever	1 to 7 days
Smallpox (variola)	8 to 12 days
Syphilis	10 to 60 days
Tetanus (lockjaw)	5 to 10 days
Trench fever	10 to 30 days
Trichinosis	5 to 10 days
Trypanosomiasis	10 days to 3 weeks, in some instances 2 to 5 years
Tsutsugamushi disease (Japanese river fever, scrub typhus fever)	7 to 18 days
Tularemia	2 to 4 days up to 10
Typhoid fever	10 to 14 days
Typhus fever	8 to 12 days
Yaws	2 to 4 weeks
Yellow fever	3 to 6 days

2 ***The Stage of Onset or Invasion:*** It may be insidious (gradual), or it may be abrupt *The insidious onset* is marked by a slowly rising temperature in which the evening exacerbations exceed the morning remissions; the prodromal symptoms become intensified and both the morning and evening temperatures reach a higher level each day until the acme or fastigium is reached. A typical example of this type of onset is typhoid fever.

The abrupt onset comes on without any or with very few prodromal symptoms; it is usually ushered in with a chill, or several chills, pallor, some cyanosis and, in children, often with convulsions. The temperature reaches its acme in several hours. This type of onset is seen in lobar pneumonia, influenza, scarlet fever, typhus fever and other febrile diseases.

3. ***The Fastigium:*** This is the stage of acme or stadium in which the temperature curve has assumed the type characteristic of the disease causing the fever. All fevers show daily variations; the maximum temperature is usually reached in the late afternoon or early evening, and the minimum in the early morning. According to the degree of the daily variations in the temperature curve, several types of fever are recognized.

A Continuous Temperature· This is one in which the diurnal variations are rarely more than 1° F. or 1.5° F. The lower level is usually found in the A. M. and the higher is reached in the P. M. Diseases characterized by continued fever are: erysipelas, kala azar, paratyphoid, pneumonia (pneumococcal), Rocky Mountain spotted fever, scarlet fever, septicemia, tuberculosis (miliary), typhoid fever, and typhus fever.

Remittent, Hectic, or Septic Temperature: This is one in which the daily oscillations are more than 2° F.; it may reach nearly, but not quite the normal line during its daily intermisisons. Diseases characterized by remittent fever are: brucellosis, epidemic meningitis, estivoautumnal malaria (falciparum), kala azar, pneumonia (atypical broncho-, virus, etc.), rheumatic fever, rheumatoid arthritis, subacute bacterial endocarditis, and tuberculosis.

The following diseases have a fever with *one remission:* dengue, measles, smallpox, virus infection (acute), and yellow fever.

Intermittent or Relapsing Fever: This is one in which the temperature reaches or goes below the normal line where it may remain for several hours or days before it again rises abruptly to its previous febrile level or a higher level. Diseases characterized by intermittent fever are· brucellosis (undulant fever), Charcot's intermittent hepatic fever, Hodgkin's disease, malaria, relapsing fever, septicemia, and tuberculosis

Recurring Fever: A return or recrudescence of fever, after the temperature had remained normal for some time, may be caused by a relapse of the previous disease, the onset of a new disease or the onset of a late complication of the original disease.

Chronic Febrile Diseases. subacute bacterial endocarditis, septicemia, tuberculosis, undulant fever, lupus erythematosus disseminatus, and periarteritis nodosa.

The Inverse Type of Temperature· It is so called when the exacerbations take place in the morning and the remissions in the evening.

Atypical or Irregular Temperature Curves: These follow no definite pattern.

4. ***The Decline of Fever:*** It may be gradual (lysis) as in typhoid fever, or it may be abrupt or sudden (crisis) as in lobar pneumonia. Occasionally there may occur a pseudocrisis, that is, the temperature falls suddenly to the near normal, but rises again within several hours. This often precedes the true crisis which is marked not only by the sudden drop of temperature to the nor-

mal, but also by the sudden amelioration of all toxic phenomena.

Subnormal Temperature: A temperature below 97° F. (36 1° C.) is considered subnormal Subnormal temperatures are found in shock, severe hemorrhage, wasting diseases, severe exhaustion, myxedema, chronic heart and lung disease with cyanosis, on exposure to intense cold, immediately preceding or during a chill, in certain types of mental disease and in those subjected to freezing. A subnormal temperature associated with a weak, rapid or unusually slow pulse is a danger signal

1. Relation of the Temperature to the Pulse Rate, Respiratory Rate and Basal Metabolic Rate

A rise in temperature of 1° F. is accompanied by the following signs:

1. The pulse rate increases from eight to ten pulse beats per minute, except in scarlet fever, septicemia, certain types of heart affections and exophthalmic goiter where the rate is proportionately faster, and in typhoid fever, meningitis, intracranial pressure, myxedema and certain myocardial changes where the rate is proportionately slower.

2. The respiratory rate is increased by about 2 to 2½ respiratory cycles per minute except in pulmonary disease when the rate is proportionately increased

3. The basal metabolic rate is increased about seven per cent, except in exophthalmic goiter where it is higher and in myxedema and nephrosis where it is proportionately lower.

2. Etiology of Fever

Fever is a symptom of disease and not a disease in itself. Diseases are classified according to their etiology; many of them, though of widely divergent etiology may, nevertheless, have several symptoms in common, and fever is often one of them. Most of the acute infections and many of the contagious diseases, though of varied etiology and symptomatology, have the common phenomena of elevated temperature. The type of temperature often varies with the kind of infective agents such as bacilli, cocci, viruses, rickettsia, spirochetes, protozoa, mycosae, and agents of unknown morphology which cause general or local infection.

Other causes of elevated temperature are the introduction of foreign protein or impurities into the blood stream; the liberation in the body of abnormal proteins such as the absorption of blood after a large hemorrhage or after an extensive surgical operation; the absorption of necrotic tissue following coronary thrombosis, pulmonary infarcts, widespread metastatic malignancy, particularly when the liver is invaded; and the absorption of pus Fever also occurs in excessive dehydration which prevents heat dissipation and in disturbance of the heat regulating centers as in certain lesions or injury to the base of the brain and the spinal cord. Occasionally no definite cause for the abnormal rise in temperature may be discernible; the unexplained fevers belong to this category

The production of bodily heat bears a definite relation to the oxygen consumed and the carbon dioxide eliminated A rise in temperature above normal may therefore be caused by the following: (1) increased heat production, (2) decreased heat dissipation, and (3) by both increased heat production and decreased heat dissipation.

Fever Caused by Increased Heat Production:

1 Infectious fevers caused by invasion of pathogenic micro-organisms

2 Postoperative or surgical fever, possibly due to the absorption of blood or other toxic materials from the injured tissues

3 Hemolysis (nonspecific fevers) caused by intravenous injections of foreign proteins, pyrogens, unmatched blood, distilled water, or other substances

4 Nervous fevers caused by injury to the central nervous system, such as trauma or hemorrhage, particularly in the neighborhood of the third ventricle, pons, medulla, and upper portion of the spinal cord

5 Fevers caused by strenuous exercise

6 Fevers caused by thyroid crisis

7 Drug fever, such as that induced by the administration of large doses of caffeine and cocaine, causing increased muscle tone, dinitrophenol causing increased tissue oxidation, and adrenalin and thyroxin in large doses which produce a high metabolic rate

Fever Due to Decreased Heat Dissipation:

1 In conditions which interfere with radiation and evaporation

2. In heat stroke, due to exposure to hot and humid air.

3 In severe dehydration (hydremia) caused by drastic cathartics, severe diarrhea, dysentery, incessant vomiting, or great loss of blood.

4 After subcutaneous injection of betatetrahydronaphthylamine or other drugs that cause marked vasodilation

5. After intravenous injections of large amounts of hypertonic solutions of saline or glucose.

6 From pronounced hemoconcentration *due to any cause.*

Fever Due to Both Increased Heat Production and Decreased Heat Elimination:

1 Infectious fevers.

2 Fevers from brain injury.

3. Fevers in malignancy

4 Fevers from blood dyscrasias due to disease or caused by drugs

5 Fevers from sun stroke.

3. Diagnosis of Fever

In addition to an abnormal rise in temperature, fever is usually accompanied by other signs such as disturbed nutrition, loss of weight, dryness of tongue, anorexia, weakness, sweats; and often by various toxic and nervous manifestations, such as headache and tremor. In prolonged or very high temperatures there may be somnolence, stupor, delirium, coma, and gastrointestinal disturbances. *The urine is usually highly* colored and scanty and there may be constipation with abdominal distention The blood count varies, depending upon the type of infection; in most of the febrile conditions, there is a leukocytosis; in some, as in typhoid fever, malaria, undulant fever, measles, and influenza, there is a moderate leukopenia The presence of leukopenia in a disease where leukocytosis is the rule is an *ominous sign. The differential leuko*cyte count is also characteristic in some infections. Blood cultures, sera reactions, agglutination tests, and examinations such as cultures of the excreta and of the spinal fluid, together with the physical signs and, in certain cases, x-ray studies will help to identify the cause of the disease in which fever is a prominent symptom

Febrile diseases of less than seven days' duration seldom require elaborate differential diagnosis. At times the diagnosis of such diseases is readily made on the evaluation of the history, the symptomatology, the physical examination and the routine laboratory examinations Quite frequently a definite diagnosis is not made in such cases and the return of the temperature to normal within a few days of its onset makes further studies unnecessary, particularly so if the patient is well and no contact cases have occurred

At the very beginning of a febrile disease in which pathognomonic signs are absent, a diagnosis cannot be made and it is not always possible to foretell whether it will be a fever of short or long duration, or whether it is the beginning of a contagious or noncontagious disease; nor can it be entirely foretold whether the disease just developing is one that requires specific therapy or not, or what the final outcome will be. Therefore, it is necessary to study each case thoroughly and not take a diagnosis "for granted" and dismiss the case as one of grippe, indigestion, sinusitis, etc., unless there are enough data to substantiate such diagnosis Whenever the diagnosis is obscure, the physician should at all times be ready to admit, at least to himself, that he is uncertain of the diagnosis and should seek such aid as may enable him to identify the illness.

In the more prolonged febrile diseases, the diagnosis of typical cases may often be made quite early in their course, this being based on the history, symptoms, physical signs and such of the laboratory procedures as suggest themselves in the particular ailment at hand Atypical cases, those in which the usual pathognomonic signs are absent or appear late in the disease, or in which the signs are masked, as well as diseases that have superficial resemblance but are of widely varied etiology, and in which the general course, symptomatology and other findings are not readily classifiable as distinct entities, require especially thorough study and the use of all the diagnostic procedures (SEE: Résumé Tables, pp. 59-59g).

III. Hyperhidrosis (Sweats)

Hyperhidrosis: Sweating is one of the methods by which heat is dissipated from the body. Insensible sweating is a constant process in health. Visible sweating in health occurs in overheating and overexertion; it may also occur in emotional states such as fright, eagerness, and elation. Hot drinks and drugs such as pilocarpine, camphor, alcohol and ipecac will induce it. Sweating also occurs in shock, in severe pain, in hyperinsulinism and other states of hypoglycemia. It is seen at the crisis of acute infectious fevers, such as pneumonia, influenza, typhus fever, etc. Irregular sweats occur in septicemia, pyemia, pyonephrosis, empyema, and where there are collections of pus or necrotic tissue, as well as in various other diseases such as malaria, dengue, miliary fever, influenza, rheumatic fever, undulant fever, bronchopneumonia, tetanus, myocarditis, gout, polyneuritis, morphinism, pulmonary tuberculosis (night sweats), exophthalmic goiter, the various neuroses, vagatonia, sympatheticotonia and in general debilitated states, particularly in those associated with low blood pressure and in certain types of adrenal tumors. Local sweats under the arms, on the palms or the feet may occur in apparently normal individuals or in those who have a sensitive nervous system. Local sweating also occurs in

facial hemiatrophy, aortic aneurysm, migraine, syringomyelia, tabes dorsalis, multiple neuritis, mediastinal abscess, rickets, and exophthalmic goiter.

IV. Anidrosis (Deficiency of Sweat)

Excessive dryness of the skin occurs in ichthyosis, scleroderma, myxedema, cretinism, diabetes insipidus, profuse diarrheas, excessive vomiting, high fevers, scurvy, diabetes mellitus, chronic interstitial nephritis, depressive psychosis, adiposis dolorosa, anorexia nervosa, and hepatic cirrhosis. Local anidrosis may occur in local vascular disease, in local nerve injuries and in local skin disease, as seen in thromboangiitis obliterans, in arteriosclerosis obliterans, in Horner's *syndrome* (*unilateral anidrosis*) and in morphea and other trophic skin lesions.

V. Rigors (Chills)

Chills consist of sudden tremors of varying extent and duration and are usually accompanied by a sensation of cold. They may be followed by fever or by a sensation of warmth and often by sweats. Chills may be caused by exposure to cold, by psychic disturbances or they may have a true clinical significance.

Chills of true clinical importance are followed by an abrupt rise in temperature and usually signify infection or trauma. They occur in the following conditions

Lobar Pneumonia (*Pneumococcic*)· The disease is often initiated with an abrupt chill.

Atypical Pneumonia (*Bronchopneumonia*) : Chilly sensations often precede the onset of the disease.

Malaria: This disease is characterized by periodic attacks of chills, fever and sweats.

Pyelitis and Pyelonephritis: *Here* chills may recur at regular or irregular intervals.

Subacute Bacterial Endocarditis: Chills usually occur with embolic phenomena and are followed by rise in temperature and sweats.

Injections into the blood stream: Foreign protein, unmatched blood, certain drugs and sera injected into the blood stream cause severe chills followed by an abrupt rise in temperature and sweats.

Puerperal Sepsis, etc.: This and septicopyemia and general blood stream infections cause chills, fever and sweats.

Acute Peritonitis, etc.: This as *well as acute osteomyelitis, erysipelas* and other acute infections and also pulmonary, renal and other suppurations are characterized by *chills. Severe chills* also occur in empyema, phlebitis, renal embolism, renal calculi, the passage of a urethral catheter, gallstone colic, empyema of the gallbladder, hepatic abscess, perineal abscess and in certain acute fevers, *e. g.*, influenza, typhus fever, variola, rheumatic fever, relapsing fever, tularemia, cerebrospinal meningitis, and in allergic shock.

Recurrent Chilliness: This condition may be found in general sepsis, and in liver and bile duct suppuration. Chilly sensations are often complained of during the menopausal stage, and in various emotional disturbances as fear, fright and psychic disturbances.

Résumé of Febrile Diseases

1. *Bacillary Infections*

Disease	Onset	Temperature Type	Duration	Pulse	Infective Organism	Leukocytes	Symptoms and Physical Signs	Laboratory Tests
Anthrax	Fairly rapid	Variable—103° to 104° F or no fever	6 to 8 days or longer May be fatal in 3 days	Rapid and weak	Bacillus anthracis	Moderate.	**Cutaneous Form** Localized furuncle on face, neck or arms, ruptures within 36 hours leaving a blackish eschar Enlargement of regional lymph glands **Pulmonary Form** Signs of bronchopneumonia **Gastrointestinal Form** Signs of severe gastric enteritis with peritonitis Large spleen	Bacilli may be found in infected tissue Inoculation of guinea pigs or mice, to be killed in 48 to 72 hours.
Bacillary Dysentery	Acute	Continuous fever with moderate remissions	Months.	Follows temperature	Dysentery bacillae of Flexner, Shiga (severe form), Sonne, and Schmitz types	Leukocytosis may be slight or moderate	Enteritis with much pus and blood in stool Colic, tenesmus and prostration.	Culture and stool examination, and serum agglutination tests.
Glanders	Fairly rapid.	Irregular—septic type	In acute form, 2 to 4 weeks, usually fatal, in chronic form, 2 to 3 years, marked by periods of remission	Rapid.	Bacillus mallei (Pfeifferella mallei)	During stage of suppuration, leukocytes 13,000 to 15,000 with 80 per cent neutrophils In later stages, leukopenia	Inflammation at site of infection, lymphangitis. Pustules in various parts of body appear between 6th to 12th day. Abscess in muscles and internal viscera with suppuration of lymphoid tissue Often diarrhea, exhaustion and death	Presence of mallei bacilli in tissue Intraperitoneal injection of male guinea pig. Characteristic growth on potato Agglutination test with antimalleus serum.
Diphtheria	Gradual.	Moderate at onset, then fairly continuous.	3 to 6 weeks	Rapid, out of proportion to temperature.	Bacillus diphtheria.	Leukocytosis	Fever, headache, malaise, sore throat, membraneous exudate on mucous surface (throat, larynx), prostration.	Positive cultures
Paratyphoid Fever	Fairly rapid.	Continuous with morning remissions of 1.5° to 2° F Termination by rapid lysis.	10 days to 2 weeks	Slow rate, faster than in typhoid fever	Bacillus paratyphosus A, B, (and probably C B. suipestifer).	Leukopenia as a rule	Resembles milder types of typhoid fever Rose colored large spots often Spleen somewhat enlarged Headache prominent	Agglutination tests for paratyphoid A & B positive about 10th day. Feces, urine and blood cultures usually positive for A and B

1. *Bacillary Infections* (Continued)

Disease	Onset	Temperature Type	Duration	Pulse	Infective Organism	Leukocytes	Symptoms and Physical Signs	Laboratory Tests
Plague	Abrupt with chill.	104° to 105° F with morning remissions. Terminates by lysis	1 to 2 weeks or longer Death may occur within 1 week	Rapid, weak, often irregular	Bacillus pestis. Transmitted from rodents to man by fleas.	20 000 to 60 000 In septicemic or pneumonic forms there may be leukopenia.	Headache, epistaxis, diarrhea delirium, bubos in bubonic form Lung consolidation in pneumonic form Bacteremia in septic form Enlarged spleen	Plague bacilli in material from bubos, and in sputum in pneumonic form. Rat or guinea pig inoculation.
Tetanus	Gradual	Moderate as a rule Fatal hyperpyrexia may occur	Days to weeks	Follows temperature	Clostridium tetani enter the body by way of punctured or perforated wound.	10 000 to 15 000	Spasm of muscles at wound restlessness irritability headache, stiff jaw and neck, trismus, sardonic grin, opisthotonus convulsions and muscle spasms collapse sweating etc, follow	Attempted bacteriologic diagnosis by culture
Tuberculosis	Gradual	In acute stages, continuous with moderate remissions In septic stage, irregular and septic In chronic stages irregular	Depends on type Months to years.	In acute, slow in chronic rapid	Tubercle bacillus	If uncomplicated, no increase.	Vary with lesions may be pulmonary glandular meningeal visceral, G U, etc	Tubercle bacilli in infected tissue or excretions Positive tuberculin tests. Guinea-pig inoculation with suspected material
Tularemia	Usually abrupt	May remain high or have daily remissions Distinct remission occurs often after 1st and 3rd days	10 to 21 days as a rule often much longer due to complications. May last several months.	Follows temperature	Bacterium tularense Transmitted by rabbits and other rodents, and by tick bites, fly bites and infected animal bites.	15 000	Headache, chills, fever, sweats, prostration, cramps delirium aching eyeballs, common ulcer formation at site of infection, swelling of regional glands The five principal types are (a) Ulceroglandular (b) Oculoglandular (c) Glandular (d) Typhoidal. (e) Visceral	Agglutination test positive in 2nd week and increased rapidity in titer Positive in 1 80 or more Skin test often positive
Typhoid Fever	Gradual-	Continuous with morning remissions Terminates by lysis.	3 to 4 weeks or longer	Slow rate, often dicrotic.	Bacillus typhosus of Eberth.	Leukopenia during the early stages and in absence of complications.	General apathy Enlarged spleen. Rose colored spots on lower chest and upper abdomen appear on 7th or 8th day Diarrhea often	Widal reaction positive in dilutions 1 50 and higher after 9th day. Blood cultures positive early Stool and urine cultures positive after second week.
Undulant Fever (Brucellosis)	Slow and insidious	May be continuous for a time, later irregular, undulating and remittent Terminates by slow lysis.	3 weeks to 18 months.	Usually slow	Brucella melitensis or Brucella abortus, Co-primus (goat), Br porcinus (hog), Br bovinus (cattle)	Leukopenia or normal count	Weakness, sweats, pains in joints and muscles. Insomnia, nervousness and headache	Skin tests often positive. Agglutination tests in 1 100 positive. Blood cultures and animal inoculation may be positive.

2. *Coccal Infections*

Disease	Onset	Temperature Type	Duration	Pulse	Infective Organism	Leukocytes	Symptoms and Physical Signs	Laboratory Tests
Erysipelas	Sudden with chill	Remittent type—terminates by lysis	7 days approximately	Follows temperature	Streptococcus erysipelas hemolyticus	Leukocytosis 12,000 to 20 000	Chill, pain at site, malaise, prostration, inflamed skin with line of demarcation	No specific test
Pneumonia (Atypical)	Gradual or sudden	Continuous or intermittent, terminates by lysis or slow crisis.	1 to 3 weeks.	Follows temperature	Virus, influenza, streptococcus, staphylococcus, mycose or other organisms	Normal or leukopenia, occasionally leukocytosis.	Upper respiratory infection chills, fever, cough, occasionally hemoptysis, dyspnea, etc, signs of small consolidation in one or both lungs	Sputum. X-rays
Pneumonia (Lobar) Pneumococcic	Sudden	Continuous 7 to 9 days, drops by crisis	Approximately 1 to 3 weeks	Follows temperature	Pneumococcus various types	Leukocytosis 20 000 to 30,000	Upper respiratory infection at times, chill, pain in chest, cough, hemoptysis, fever, prostration, dyspnea, signs of lung consolidation	Pneumococcus in sputum Bacteremia X-ray
Septicemia	Sudden with chills	Usually high in acute cases, and may be continuous in staphylococcus infection Intermittent in gonococcus and colon infection. Irregular in any type of streptococcus infection	Days to weeks	Proportionate to the temperature	Streptococci staphylococci, gonococci colon bacilli and other organisms	Leukocytosis or normal leukocyte count with normal polynuclears	Depends upon site of infection, usually headache, emboli, metastatic abscess as skin rashes and other local lesions, and general signs of infection Spleen is enlarged	Blood cultures
Streptococcus Sore Throat	Sudden	101° to 104° F Continuous at onset, later irregular Terminates by rapid lysis	3 to 7 days or longer	Rapid, follows temperature	Streptococcus	Leukocytosis 14,000 to 18 000	Malaise fever, prostration, difficulty in swallowing, swollen, edematous membranes of throat, tonsils, etc	Culture of throat
Subacute Bacterial Endocarditis	Insidious	Remittent temperature curve	Weeks to months	Follows temperature	Strep viridans	12,000 to 18,000	Fatigability, anorexia, general malaise, euphoria, chronic valvulitis, embolic phenomena, splenomegaly	Finally—a positive blood culture

3. *Virus Infections*

Disease	Onset	Temperature Type	Duration	Pulse	Infective Organism	Leukocytes	Symptoms and Physical Signs	Laboratory Tests
Dengue	Sudden	Continuous with remission after 24 to 36 hours and a secondary rise to previous level	7 to 10 days.	Rapid during first stage, slow during second stage	Virus transmitted by the Aedes mosquito	Leukopenia 1200 to 2000	Severe pain in head eyes, limbs and back. During second stage there is a scarlatinal or measles rash over body	Reduction in granulocytes and lymphocytes

3. *Virus Infections* (Continued)

Disease	Onset	Temperature Type	Duration	Pulse	Infective Organism	Leukocytes	Symptoms and Physical Signs	Laboratory Tests
Influenza	Sudden	Mild form Continuous 100° to 104° F Terminates by crisis or rapid lysis Severe form Continuous *103° to 106° F* Terminates by crisis or rapid lysis	Mild form 3 to 5 days Severe form 7 to 14 days	80 to 100 Slow in comparison to temperature	Virus influenzae types A, B or other suspected type Secondary invaders Haemophilus influenzae, pneumococcus, Streptococcus haemolyticus, S viridans, staphylococcus, meningococcus, Friedlander's bacillus, and other organisms	Normal, with relative lymphocytosis at onset, later leukopenia	*Uncomplicated cases* Severe headache, backache, generalized pains, prostration, apathy, gastrointestinal disturbance, upper respiratory involvement. Complications Pneumonia, bronchiectasis, pulmonary fibrosis May be epidemic or pandemic	*Leukopenia, relative lymphocytosis*, occasional high erythrocyte count Isolation of virus whenever possible
Psittacosis	Sudden	Elevated — irregular 101° to 103° F	2 to 4 weeks	Slow	Virus—transmitted by parrot or other infected birds.	Leukocytosis.	Malaise chill, mild G I complaints, central pneumonic involvement.	Mice inoculation with patient's sputum may cause characteristic lesion in liver and spleen. Agglutination test
Rabies (Hydrophobia)	Gradual	Continuous often 104° to 105° F. Before death it may rise to *106° to 109° F*	Death occurs within 3 or 4 days.	Rapid.	Virus.	10 000 to 20 000, also high erythrocyte count	Husky voice, trismus, inability to swallow, spasm of muscles of deglutition and respiration *hyperesthesia and convulsions*	Negri bodies in large cells of central nervous system of an infected animal, Infected ani*mal dies within a few days.* Rabbit inoculation.
Smallpox (Variola)	Sudden.	1st stage, continuous —103° to 104° F, 2nd stage, normal or near normal temperature, 3rd or pustular stage, temperature rises to 102° to 105° F, lasting from 3 to 12 days Terminates by crisis or lysis	1st stage — 3 days, 2nd stage—3 days, 3rd stage—3 to 12 days.	Proportionate to temperature.	Virus	1st stage — 10,000 to 15 000, 2nd stage—8000 to 9000, 3rd stage—20 000 to 30,000 In overwhelming infection there may be leukopenia.	Headache, pain in extremities and in lumbar region, preeruptive rash. Erup on undergoes 3 distin t stages—macular, vesicular, and pustular The eruption is confluent on face, forehead, wrists, arms, trunk and feet.	Culture of virus on rabbit's cornea. Complement fixation with fluid from vesicle and in specific serum. Intradermal rabbit test
Yellow Fever	Sudden.	Continuous 102° to 105° F during the first 3 days followed by a remission of 12 to 24 hours and a secondary rise to previous level. In nonfatal cases temperature falls by lysis	6 to 12 days	Very slow, out of proportion to temperature Pulse rate may become slower while temperature is still high or is rising (Faget's Sign)	Filtrable virus in the body fluids transmitted by Aedes aegypti mosquito	Leukopenia is the rule In fatal cases there may be leukocytosis	Flushed face, injected eyes, tender epigastrium, black vomit, jaundice appears on 3rd day and is progressive Bleeding from mucous surfaces. Anuria	Van den Bergh direct reaction. Icterus index is high. Liver function tests indicate liver necrosis.

4. *Contagious Diseases of Childhood*

Disease	Onset	Temperature Type	Duration	Pulse	Infective Organism	Leukocytes	Symptoms and Physical Signs	Laboratory Tests
Chickenpox (Varicella)	Abrupt.	Slight if any	1 to 2 weeks	Follows temperature	Virus (?)	Normal or mild leukocytosis.	Maculopapulovesicular pustular rash in all stages on all parts of body and extremities.	Nonspecific.

4. *Contagious Diseases of Childhood* (Continued)

Disease	Onset	Temperature Type	Duration	Pulse	Infective Organism	Leukocytes	Symptoms and Physical Signs	Laboratory Tests
Measles (Rubeola)	Gradual	High at onset with remission after 2 to 3 days and then fall by lysis.	2 to 3 weeks.	Follows temperature	Virus (?)	Leukopenia with lymphocytosis	Catarrhal symptoms, cough, Koplik's spots, confluent maculopapular rash face to trunk and extremities by 3rd or 4th day.	Nonspecific
Scarlet Fever (Scarlatina)	Abrupt	High for first few days, falls by lysis by end of week	2 to 4 weeks	More rapid than temperature and wiry	Hemolytic streptococcus.	Leukocytosis with great increase of polymorphonuclears. During convalescence there is an increase in the eosinophils	Chill, convulsions, vomiting, sore throat, punctate rash in throat Rash from head down in 24 hours Face exempt Circumoral pallor, Strawberry tongue Desquamation 10 to 14 days later. (Complications frequent)	Dick test positive first few days Shultz-Charlton reaction good diagnostic aid.

5. *Rickettsia Infections*

Disease	Onset	Temperature Type	Duration	Pulse	Infective Organism	Leukocytes	Symptoms and Physical Signs	Laboratory Tests
Rocky Mountain Spotted Fever	Abrupt with a chill.	Continuous 103° to 104° F. for approximately 2 weeks with morning remissions, lysis during 3rd week.	Approximately 21 days.	Bounding at first, becomes rapid out of proportion to temperature	Microorganism of Rickettsia group. Transmitted by infected ticks—Dermacentor andersoni	Mild—12,000 Increase in mononuclears Decrease in eosinophils	Chill, headache, severe muscle and joint pain, toxemia, enlarged spleen, characteristic macular rash usually on 3rd day, abdomen exempt or sparse, extremities to trunk	Weil-Felix serum reaction with proteus bacillus X19 is often positive.
Trench Fever	Abrupt	Three types (1) Moderate, 5 to 7 days and followed by remission and short secondary rise; (2) Continuous fever for 6 to 7 weeks, (3) Periods of fever followed by alternate afebrile and *febrile periods for* weeks	Variable	Follows temperature	Rickettsia quintana or pediculi Transmitted by infected lice	Moderate as a rule, normal w b c. or leukopenia may occur.	Prostration, headache, muscle pains usually of legs, enlarged spleen, transient macular rash for hours to 2 days Tender shins during the night are characteristic	Weil-Felix may be positive In trench fever as in typhus fever the Wassermann reaction is often positive before the crisis.
Tsutsugamushi Disease (Japanese River Fever)	Abrupt	Continuous high fever for 2 to 3 weeks, declining by rapid lysis	14 to 21 days	Follows temperature	Rickettsia group Transmitted by bite of infected trombidium mite	Leukopenia at height of disease	Headache, malaise, local ulcer with enlargement of regional glands, macular rash at end of 1st week lasting 3 to 4 days, toxemia in severe cases, splenomegaly	Agglutination reaction negative for B proteus X19, but generally positive for the K strain near end of fever
Typhus Fever	*Abrupt*	*High febrile level with* slight diurnal variations for about 14 days. Terminates by crisis or rapid lysis	*Approximately* 14 days.	*Follows temperature.*	*Rickettsia* prowazeki, transmitted by infected body lice.	*Mild increase, 12,000*	*Chill, febrile* reaction, anxious face, delirium at times, muscular pains Enlarged spleen during first week Bronchitis Rash on 4th to 5th day, face exempt, trunk and extremities undergo changes Mild conjunctivitis	Weil-Felix agglutination. Proteus bacillus X19 will agglutinate in dilution 1 100 to 1 500 of the patient's serum.

6. Spirochetal Infections

Disease	Onset	Temperature Type	Duration	Pulse	Infective Organism	Leukocytes	Symptoms and Physical Signs	Laboratory Tests
Epidemic Jaundice (Weil's Disease)	Abrupt with chills	Irregular Terminates by lysis or crisis.	7 to 10 days or longer Relapses may occur	Rapid	Leptospira icterohemorrhagica (icterohaemorrhagiae)	Moderate leukocytosis.	Jaundice, large spleen and liver, hemorrhage from mucous surfaces, nausea, vomiting, weakness and generalized pain	Leptospira in the urine and blood Guinea pig inoculation.
Rat-Bite Fever	Abrupt.	Irregular, may reach 104° F Relapses common	Days to weeks	Rapid	Treponema morsus muris (Sprillum minus, and a streptobacillus)	Moderate leukocytosis	Inflammatory lesion at site of bite Enlarged regional lymph glands.	Spirochete may be found in the lesions, and in the blood of inoculated mice.
Relapsing Fever (Tick Fever)	Sudden	Intermittent Temperature 103° to 105° F for 5 days. Sudden decline Relapses occur after several days of normal temperature	Weeks.	Follows temperature	Spirochete of genus Borrelia (S. recurrentis) Transmitted by infected ticks and lice	Moderate leukocytosis.	Headache, fever, malaise, abdominal pain, constipation nausea, vomiting, enlarged liver and spleen — relapse after afebrile period of 1 to 2 weeks.	Spirochetes demonstrated in blood during febrile periods. Positive Rieckenberg's or Adhesive test.
Syphilis	Gradual	May be afebrile, low continuous fever, intermittent, or remittent	Years.	Proportionate to temperature In heart complications may be very slow or very fast	Treponema pallidum.	Not characteristic.	Depend upon stage of disease and site of involvement Any tissue or organ may be involved.	Serologic Test (Wassermann, Kahn, Kline and other tests) Presence of spirilla in tissue juices and in tissues as seen in dark field preparations.
Vincent's Angina	Gradual	Mild elevation.	Weeks to years.	Follows temperature	Primarily unknown, secondarily fusiform and spirochetes.	Moderate leukocytosis.	Ulceration and sores of membranes of gums, mouth, etc.	Culture from mouth and gum lesions will show spirochetes.
Yaws (See p 143) Stage of nasal ulceration and destruction is known as Gangosa (see p 149)	Gradual	Follows stage with amount of involvement accounting for temperature, irregular, intermittent	Months.	Follows temperature	Treponema pertenue	No leukocytosis, occasionally increase of monocytes.	Three stages are recognized (1) Mother yaw-papula encrusted lesion with granulating base. Extragenital—regional adenopathy (2) Generalized papular eruption which develops into characteristic yellow crusts. Removal shows granulating surface (3) Tertiary lesions are nodular or ulcerative. May involve skin or bones. Internal viscera not involved.	Wassermann plus 4 Treponema present in lesion

7. *Protozoal Infections*

Disease	Onset	Temperature Type	Duration	Pulse	Infective Organism	Leukocytes	Symptoms and Physical Signs	Laboratory Tests
Amebiasis	Acute or gradual	Irregular type, subfebrile or low febrile	Days to weeks	Follows clinical state	Endamoeba histolytica	Eosinophilic increase with slight increase in white blood count	Constipation and diarrhea, colic, pain in (R L Q,) anorexia, headache, asthenia, mucus and blood in stool	Demonstration of organism in feces
Kala-azar	Insidious	Remittent fever, often a double rise in 24 hours	Weeks to years	Follows temperature	Leishmania donovani (Protozoan parasite)	Leukopenia 2000 to 4000 Relative lymphocytosis and monocytosis	Irregular fever, emaciation, dysentery, cachexia, greatly enlarged spleen Marked anemia	Demonstration of Leishmania donovani in smears of peripheral blood
Malaria	Abrupt	Intermittent, remittent, continued, depending upon the type of protozoa. Occurs in cycles of chills, fever and sweats	Months, modified by treatment	Rapid, irregular	Malarial parasites Transmitted by mosquitoes	Leukopenia with increase in large mononuclears	General malaise, headache, chill followed by high temperature Severe headache, muscle pain, occasional delirium or semi-coma. Sweating follows Enlarged spleen Quotidian Q 24 hrs Tertian Q 48 hrs Quartian Q 72 hrs Estivo-autumnal Q 40 to 48 hrs Infection with several strains may give daily or bi-daily paroxysms	Demonstration of Plasmodium malariae in blood Therapeutic test with quinine

8. *Meningites (See: pp. 877-880)*

Disease	Onset	Temperature Type	Duration	Pulse	Infective Organism	Leukocytes	Symptoms and Physical Signs	Laboratory Tests
Cerebrospinal Meningitis	Abrupt	Irregular Terminates by lysis.	3 days to weeks	Follows temperature or may be slow	Meningococcus of several types	20 000 to 30,000	Headache, chill, vomiting, delirium, stiff neck, Kernig's sign, occasional rash, coryza, hyperesthesia	Spinal tap shows 1 Increased pressure 2 Turbid fluid 3. Meningococci 4 Increased cell count (polys) 5 Decreased sugar Blood culture may be positive
Tuberculous Meningitis	Gradual	Irregular Terminates by lysis	Hours to days	Follows temperature	Tb bacillus	Moderate increase	Headache, rigid neck, Kernig's sign, delirium, etc	Spinal tap shows 1 Increased pressure 2 Early—slightly cloudy (meniscus), later—cloudy 3 Lymphocytosis 4 Low chlorides to below 600 mg per 100 cc 5 Tubercle bacilli 6 Positive Levenson test

Other types of meningitis are diagnosed by symptoms common to all types of meningitis and by the specific organisms found in the spinal fluid

9. *Diseases of Doubtful Origin*

Disease	Onset	Temperature Type	Duration	Pulse	Infective Organism	Leukocytes	Symptoms and Physical Signs	Laboratory Tests
Agranulocytic Angina	Abrupt with chills	High with remissions.	Weeks. Often fatal	Follows temperature	Unknown—may follow chemical poison, severe fever or irradiation	Leukopenia with practically complete loss of polymorphonuclears.	Chills, fever prostration, sore throat with ulcers, occasionally jaundice	Blood smears will show rapid disappearance of granulocytes and pronounced leukopenia,
Hodgkin's Disease	Insidious	Varied — moderate, septic, remittently febrile and afebrile	Up to a few yrs.	Follows temperature	Unknown	Mild increase	Weakness, weight loss, dyspnea, chills, fever, painless enlarged glands, splenomegaly, late	Biopsy of gland will show Dorothy Reed cells.
Infectious Mononucleosis	Acute	103° F., 2 to 5 days, followed usually by lysis	2 to 3 weeks.	Follows temperature	Unknown.	3000 to 35 000, often 90% monocytes.	Sore throat, headache, malaise, marked adenopathy, splenomegaly, conjunctivitis.	Heterophile antibody test is positive in dilutions over 1 to 160 after the second week. Wassermann reaction may be positive.
Periarteritis Nodosa	Insidious.	Remittent, ranging from 100° to 102° F	Months.	Rapid.	Unknown	20,000 to 50 000	Severe abdominal pain, bronchitis, digestive disorders signs of nephritis, apathy, nodular swelling along arteries and subcutaneously At times they are not apparent	Biopsy
Rheumatic Fever (Acute)	Sudden or insidious.	102° to 105° F with remissions and acid sweats.	Weeks to months	Rapid. Out of proportion to temperature	Doubtful. Suspected—streptococcus hemolyticus.	Leukocytosis, 10,000 to 25,000	Varied—upper respiratory infection plus anemia, malaise, migrating polyarthritis, tonsillitis, malnutrition, etc. Cardiac valvulitis. In children, subcutaneous nodules.	None specific. Electrocardiogram. Sedimentation time is rapid.

10. *Miscellaneous Causes*

Disease	Onset	Temperature Type	Duration	Pulse	Infective Organism	Leukocytes	Symptoms and Physical Signs	Laboratory Tests
Encephalitis Lethargica	Gradual or Abrupt.	Irregular Terminates by lysis.	Hours to weeks to years.	Follows temperature.	Virus.	Moderate increase.	Headache, diplopia, lethargy, palsies, neck rigidity, Kernig's sign.	Spinal tap 1. Lymphocytes. 2. Increased sugar. 3. Chlorides not reduced.
Sprue	Insidious	Slight or none with remissions and exacerbations	Often years.	Follows temperature	Fungus, vitamin deficiency	Slight increase.	G. I. unrest—epigastric distress, flatulence, diarrhea and constipation, red tongue with vesicular ulcerations at edge Characteristic stool, with fat, bubbles, white or putty color Asthenia and depression.	Macrocytosis, increased color index, anisocytosis, poikilocytosis.
Trichinosis	Abrupt	Moderate first week, continuous at 103° to 105° F., 2nd to 3rd weeks gradual lysis. Fever may be prolonged and remittent	4 to 6 weeks	Follows temperature	Trichinella spiralis.	Leukocytosis plus high eosinophilia.	Onset with G. I. symptoms, then profound myositis and then possibly edema, cachexia, delirium, coma, dyspnea.	Trichinella larvae in stool or centrifuged blood. Muscle biopsy may show larvae. Skin test with antigen derived from trichinella larvae.

11. *Diseases Transmitted by Anthropods*

Disease	Transmitting Arthropod	Infective Organism
African Trypanosomiasis (Sleeping Sickness)	Tsetse flies (Glossina palpalis)	Trypanosoma gambiense, T. rhodesiense
Chagas' Disease (South American Trypanosomiasis)	Reduviid (cone-nosed) bugs (Triatoma, Panstrongylus), kissing bugs	Trypanosoma cruzi
Cutaneous Leishmaniasis (Forest Yaws, Oriental Sore)	Species of sand flies (Phlebotomus papatasii)	Leishmania tropica
Dengue (Breakbone Fever)	Mosquitoes (Aedes aegypti, A. albopictus)	Filtrable virus
Encephalomyelitis (Epidemic) (Japanese, Equine, St. Louis)	Mosquitoes (Aedes)	Virus
Encephalitis (Russian—Spring, Summer)	Ticks	Virus
Filariasis (Bancroft's)	Mosquitoes of many species (Aedes, Anopheles, Culex, Mansonia)	Wucheria bancrofti (Filaria bancrofti)
Kala-Azar	Sand flies (Phlebotomus)	Leishmania donovani
Loiasis	Tabanid flies (Chrysops dimidiata, C. silacea) (Mang flies)	Parasitic filarial worm (Loa loa)
Malaria	Mosquitoes (Anopheles)	Plasmodium vivax, P. ovale, P. malariae, P. falciparum.
Mucocutaneous Leishmaniasis (Uta Espundia)	Sand flies	Leishmania braziliensis
Onchocerciasis	Black flies (Simulium)	Onchocerca volvulus
Oroya Fever (Carrión's Disease)	Sand flies (Phlebotomus noguchi, P. verrucarum)	Bartonella bacilliformis
Pappataci Fever	Sand flies (Phlebotomus papatasii)	Filtrable virus
Plague (Bubonic)	Rodent fleas (Xenopsylla cheopis).	Pasteurella pestis
Relapsing Fever (Epidemic)	Body louse (Pediculus humanus)	Spirochaeta (Borrelia) recurrentis
Relapsing Fever (Endemic)	Soft tick (Ornithodoros)	Spirochaeta (Borrelia) duttoni
Rocky Mountain and Related Fevers (Tick Typhus)	Hard ticks (Dermacentor andersoni and others)	Rickettsia ricketsi
Tsutsugamushi (Japanese River Fever)	Red mites (chiggers) (Trombidium)	Rickettsia orientalis
Tularemia	Hard ticks (Dermacentor andersoni, D. variabilis), deer fly (Chrysops discalis)	Pasteurella tularensis
Typhus Fever (Epidemic, Classical)	Body louse (Pediculus humanus)	Rickettsia prowazeki
Typhus Fever (Endemic, Brill's Disease)	Rodent fleas (Xenopsylla cheopis)	Rickettsia mooseri
Yellow Fever	Mosquitoes (chiefly Aedes aegypti)	Filtrable virus

CHAPTER III

Alteration of the Special Senses

Patients may complain of some alteration in any of the special senses, *i. e.*, in touch, vision, hearing, smell and taste. There also may be a disturbance in the perception of some of the general sensations, such as heat, cold and pain. These sensations may be intensified, lost or perverted.

I. Touch

In certain nervous conditions, tactile sense may be absent, distorted or hyperactive, these abnormal sensations are known as anesthesia, paresthesia, and hyperesthesia.

Anesthesia: Local anesthesia of various parts of the body may be caused by injury to a sensory nerve, multiple neuritis (except lead), multiple sclerosis, spinal cord tumor or trauma, transverse myelitis, syringomyelia, cerebral tumor in the sensory area, and may occur in tabes dorsalis, leprosy, occasionally in herpes zoster, and in various affections of the sensory nerves and spinal cord. The affected part may be anesthetic to pain, heat, cold or to stereognosis (recognizing objects).

Paresthesia: This denotes perverted sensation. It is found in the various *neuroses, pernicious anemia, arterio*sclerosis, Raynaud's disease, endarteritis obliterans, acroparesthesia, interference in the circulation in a limb, frost bites, and in the various diseases of the sensory nerves, spinal cord or sensory portions of the brain that cause local anesthesia. The sensations perceived may be tingling, insect crawling, itching, smarting or burning. *Meroparesthesia* denotes alteration of the tactile sense in the extremities.

Hyperesthesia: Acute sensitivity of the skin to light stroking, pain, heat, cold, *light, actinic rays or other irritating* substances may be found in so-called sensitive skins, and in the functional neuroses, trigeminal neuralgia, neuritis, herpes zoster, migraine, peripheral neuritis, tabes dorsalis, subacute combined degeneration of the cord, acute myelitis, cerebrospinal meningitis and nerve injury. In the *thalamic syndrome* (hemiplegia dolorosa) there may be hypersensitivity to pain and temperature on one side with anesthesia on the contralateral side. There is loss of osseous sense, astereognosis, paroxysmal pain and involuntary movements on the affected side.

II. Vision[1] (Sight)

Alteration of vision is a fairly common complaint; it may occur in one or in both eyes. Sight may become affected because of disease of the various structures of the eye, the optic nerve, the optic center in the brain, and because of conditions which directly affect the eye structures or the brain.

Vision may be altered in three ways: (1) Increased vision; (2) diminished and absent vision, and (3) perverted vision.

1. *Increased Vision*

Hyperopia, or farsightedness, is usually due to some peculiarity of the eye; it may be due to the inability of parallel rays to focus on the retina, to insuf-

[1] SEE ALSO *Diseases of the Eyes* (pp. 171-182)

ficient convexity of the refracting surfaces or to shortness of the anteroposterior axis of the eye so that the focus falls beyond the retina.

Presbyopia: Farsightedness of the aged is due to loss of power of accommodation because of diminished elasticity of the crystalline lens, so that the near point of vision is removed farther from the eye.

2. *Diminished Vision*

Meropia: This is partial blindness or diminished vision

Myopia: This is a condition of shortsightedness, the parallel rays of light being focused in front of the retina.

Amblyopia: Defective vision or dimness of vision may be of various degrees; it may be unilateral or bilateral and may be due to ocular and to extraocular causes Ocular causes are. Eyestrain (asthenopia); astigmatism; myopia, presbyopia; acute conjunctivitis; interstitial keratitis; corneal opacities, disease of the cornea, the iris or the retina; opacities of the crystalline lines; cataract; sympathetic ophthalmia, tumors of the eye; glaucoma and congenital amblyopia; also diseases of the optic nerve as in optic neuritis, optic atrophy and retrobulbar neuritis, and traumatism to the eyes or to the optic nerve

Extraocular causes are: Reflex, from intestinal diseases; poisoning by wood alcohol, arsenic, mercury, bromides, cannabis indica, belladonna, opium, tobacco, and various other toxic agents, tumors of the brain, postdiphtheritic paralysis; hereditary cerebellar ataxia; thrombosis of the central vein; Raynaud's disease; leontiasis ossea, and hysteria.

Amaurosis: Blindness, complete or total, may be transient or permanent and may be due to diseases of the eyes or the optic nerve, or to extraocular conditions. Diseases of the eyes responsible for blindness are injuries to the orbit, eyeball or the various structures of the eye, such as may be seen in gonorrheal ophthalmia, panophthalmitis, suppurative iridochoroiditis and iridocyclitis, glaucoma, sympathetic ophthalmia, and cataract. Blindness caused by disease of the optic tract and nerve follows chronic retrobular neuritis, tumors of the optic nerve or optic tract and compression of the optic tract or nerve by cerebellar tumor, cerebral hemorrhage and cerebral embolus. Extraocular causes for blindness are amaurotic familial idiocy and toxic causes, such as uremia, diabetes, and poisoning by quinine or quinine derivates, wood alcohol, cannabis indica, belladonna, bromides and some of the coal tar products. Total blindness, which is usually temporary but occasionally permanent, occurs in severe anemia of the brain, in rapid and copious internal or external hemorrhage, during pregnancy, in snow blindness, in exposure to superbrilliant light, in lightning stroke, and in hysteria and malingering.

Nyctalopia: Night blindness may be a congenital condition; it is noted in retinitis pigmentosa and in Laurence-Biedl syndrome. It also occurs as a result of secondary atrophy of the optic nerve. Delayed dark adaptation is noted in vitamin A deficiency and in degenerative changes of the crystalline lens.

Hemeralopia: In day blindness the sight is poor in sunlight and in good illumination, but good at dusk, twilight and in poor illumination. This is noted in albinism, in retinitis with central scotoma, in toxic amblyopia, in coloboma of the iris and choroid, in opacity of the

crystalline lens or cornea, and in conjunctivitis with photophobia.

Perverted Vision: This classification includes various abnormalities in the appearance of objects, or of color. Objects may appear as double, halved, or distorted as to size and shape; or there may be changes in the perception of color. Excessive or nonexisting colors may be perceived or there may be partial blocking out of color or of sight.

Diplopia: Double vision, when looking with both eyes, is known as *binocular diplopia.* This occurs when both eyes are not in focus because of errors of refraction or accommodation. It may be found in disease of the eyeballs; in affections of the cranial nerves; in disease of the cerebellum, cerebrum or other parts of the brain and the meninges; and in conditions apparently unrelated to the eyes. In double vision two objects are seen instead of the existing one, each eye does not simultaneously reflect the same image on corresponding points of the two retinas. The images as seen are not uniformly distinct nor are they always on the same plane. The more distinctly appearing object is the true object, and is seen with the normal eye.

Homonomous Diplopia In this condition the false image is on the side of the deviating eye; this is associated with convergent squint.

Crossed Diplopia: In this condition the false image is on the side of the normal eye; this is found in divergent squint.

The false image appears above the true image in *paralysis of an elevator muscle,* and it appears on a lower plane in *paralysis of a depressor muscle.*

True diplopia is caused by paralysis of the ocular muscles. Functional diplopia may be seen in ordinary concomitant strabismus or cross-eyes. To differentiate the true from the functional diplopia a red lens is placed before one eye and a light is held about 10 feet in front of the eyes and moved in various positions. In paralysis of the ocular muscles two lights will be seen in relative positions, while in strabismus only one light is seen.

Conditions in Which Diplopia is a Symptom. Diplopia occurs during the early stages of encephalitis lethargica; in cerebrospinal meningitis and tuberculous meningitis because of paralysis of the oculomotor nerve; in myasthenia gravis because of weakness of the external rectus muscle; in acute alcoholism, in asthenopia (muscle imbalance) due to eyestrain, and in ophthalmoplegic migraine. In paralysis of the following cranial nerves, diplopia is due to muscle imbalance: Third nerve, because it is the motor of the eye muscles; fourth nerve, causing paralysis of the superior oblique muscle, and the sixth nerve because it produces paralysis of the external rectus muscle. Various diseases of the brain and spinal cord causing diplopia are: Cerebellar and cerebral tumors involving some of the cranial nerves; cerebral syphilis; general paresis; locomotor ataxia (tabes dorsalis), and multiple sclerosis. Diseases of the orbit which may cause displacement of the eyeball will also cause diplopia as seen in orbital cellulitis, hemorrhage, and orbital tumors. Other causes for double vision are postdiphtheritic paralysis, symblepharon, and unilateral exophthalmus or enthophthalmus.

Double vision in one eye (*monocular diplopia*) may occur in astigmatism, cerebral tumor, cataract, partial dislocation of the crystalline lens, double pupil and hysterical amblyopia.

Hemianopsia: Half vision may occur in one eye when there is a lesion of the retina, disc, or one optic nerve Hemianopsia occurring in both eyes of which the patient has usually no knowledge until tested, occurs in tumors of the optic tract, optic nerve, optic chiasm, the pituitary or pineal bodies, and tumor, abscess or other lesions of the cerebrum and cerebellum as well as in hysteria and migraine.

Hemianopsia is classified according to the parts of the eyes that show blindness, and this also indicates the position of the lesion. If blindness affects one eye or if both eyes are affected, but the blindness is not symmetrical, the lesion is in one or both optic nerves.

Homonomous Hemianopsia: The blindness is in the corresponding lateral halves of both eyes; that is, on the nasal side of one eye and on the temporal side of the other. The lesion causing this is located above the optic chiasm, and on the opposite side of the blind field.

Heteronymous or Heterolateral Hemianopsia· The blindness in this condition is on the opposite lateral halves of the visual fields, and is either bitemporal or binasal The lesion in the bitemporal type is at the central part of the optic chiasm before crossing

Wernick's Law: When a thin pencil of light thrown upon either the blind or seeing side of the retina causes contraction of the pupil, it indicates that the lesion is back of the primary optic centers. When the pupil does not contract as the light strikes the blind side, but contracts as it strikes the seeing side, it indicates that the lesion is in front of the primary optic centers.

Scotomata: Seeing dark spots before the eyes where they do not exist may be functional or organic.

Functional Scotomata: This is described by patients as grayish or dark shadows of various sizes and shapes; usually dots, lines, globules and rings that contract and expand; or dark spots may seem to persist as shapeless areas which move with changes of position of the eyes. Occasionally these appear as fly specks fleeting before the eyes (*muscae volitantes*). Scotomata are generally found in digestive disturbances, refractive errors, eyestrain and when looking intently at bright or dazzling objects as the sun, high voltage flashes or brilliant reflections. It may also occur in migraine, and in some of the neuroses; also in diabetes mellitus, lead poisoning, uremia, and severe anemia.

Organic scotomata appear in various diseases of the eye, such as vitreous and corneal opacities, cataract, glaucoma, disease of the retina, the choroid, and the optic nerve. Tumors of the pituitary gland or brain tumors causing optic neuritis or chocked disc may cause ring-shaped scotomata that may appear during central or lateral vision.

Chromatopsia (*Colored vision*): Various colors of the rainbow may be perceived when they are nonexistent. Sparks may be seen in head injuries

Red color is perceived when the pupils are dilated; when looking at brilliant lights; in cataracts; in hemorrhage in the retina or into the vitreous. In snow blindness and in tobacco scotomata the color observed may be *red* or *green*. The expression of "seeing red" when alluding to extreme anger is a figure of speech and not a fact. *Green color* is perceived in wounds of the cornea, tabes dorsalis and at times in tobacco scotomata. *Yellow vision* occurs in jaundice, and in poisoning by santonin, picric acid,

cannabis indica, amyl nitrite, digitalis, and quinine. *Blue vision* occurs in alcoholism, and *violet light* is seen during recovery from santonin poisoning. In hysteria the perception of colors or their combinations and brilliancy depends upon the imaginative skill of the sufferer. Rays of various colors, hues and lengths are at times observed by the blind or the partially blind.

Achromatopsia *(Color blindness)* Color blindness may be congenital or acquired. *Congenital color blindness* occurring in otherwise normal individuals is more frequently met with among males than females. There is usually a lack of perception of red, green or blue. (There are various standard tests for color blindness.) *Acquired color blindness* is caused by disease of the eyes such as retinitis, retrobulbar neuritis, optic atrophy, cataract, toxic amblyopia, optic neuritis; and occurs in certain toxic conditions as in poisoning by lead, salicylates, quinine, ergot, and carbon bisulfate; also in diabetes mellitus, uremia, arteriosclerosis, multiple sclerosis, epilepsy, hysteria and some of the psychoses.

Photophobia *(Intolerance to light)*: This occurs as a common symptom in many of the eye diseases, in acute febrile conditions, in nervous diseases, and in toxic states.

Eye diseases causing photophobia are: Eyestrain; astigmatism; hypermetropia; conjunctivitis due to any cause; sympathetic ophthalmia; albinism; interstitial keratitis; ulcers of the cornea; iritis, and retinitis.

Acute febrile diseases causing photophobia are those associated with conjunctivitis like measles, typhus fever, smallpox, etc.; and those in which conjunctivitis is absent such as tuberculous and meningococcic meningitis, acute encephalitis, pachymeningitis, tetanus, etc.

Nervous diseases causing intolerance to light are: Encephalitis lethargica; cerebral tumors; the neuroses; migraine, and trigeminal neuralgia (tic douloureux).

Toxic states due to quinine, belladonna, and other mydriatics, alcoholism, allergic reactions, and severe headaches frequently cause photophobia.

III. Hearing

Hearing may become defective, superacute or perverted.

Defective Hearing: This may range from mild deficiency to various degrees of deafness. It may occur in one or both ears. *Partial deafness* may be due to impacted cerumin, acute and chronic otitis media, inflammation or obstruction of the eustachian tube, otosclerosis, labyrinthitis, and disease of the various structures of the ears, auditory nerves, and the temporal bones. Among other causes are adenoids, Ménière's disease, some brain tumors, hemorrhage, and various toxic states resulting from the use of quinine and salicylates, as well as nephritis, and arteriosclerosis. It often occurs during certain febrile diseases as in typhoid fever, pneumonia, etc. During health it may occur in those working in boiler factories or among other deafening noises. *Complete deafness* is found in deaf mutes, cretins, and in those who have lost bone conduction, have auditory nerve degeneration, or have frontal lobe tumor causing auditory aphasia.

Hyperacusia: Heightened hearing may occur in irritation or stimulation of the auditory apparatus or in hypersensitivity of the nervous system. In most instances, the individual's hearing range for normal sounds is not pathologically

accentuated but ordinary noises seem to be intolerably intensified, or there may be a supersensitiveness to particular noises or to certain sounds.

Tinnitus Aurium: Ringing in the ears is a subjective phenomenon found among many neurotics, and in those who have irritable conditions of the auditory nerve. It is also found in association with partial deafness due to middle ear disease, eustachian tube obstruction, otosclerosis, obstruction of the ear canal, or to nasal obstruction. Tinnitus is a common complaint in arteriosclerosis, in severe anemia, polycythemia, in Ménière's disease, in mountain sickness, in nephritis with hypertension, in vertigo, just before fainting, in the various neuroses, and in some of the brain affections Tinnitus may be produced by overdoses of quinine and salicylates.

IV. Smell

The sense of smell may be weakened or lost; it may be heightened; or it may be perverted (SEE pp 187 and 855).

Anosmia: Loss of sense of smell occurs in acute and chronic diseases of the nose; in disease of the frontal, ethmoidal and antral sinuses; in acute and atrophic rhinitis, in tumors occurring in the frontal or parietal lobes, and in other lesions that exert pressure upon the olfactory pathway

Hyperosmia: *Heightened* sense of smell is seldom due to disease of the olfactory apparatus Some individuals are normally more acutely sensitive to odors than are others, it may exist as an allergic phenomenon towards certain objects, gases, or scents Hyperosmia is also found among neurotics, in hysteria and in the insane.

Parosmia: Perversion of the sense of smell is of two types: One in which there is a perversion of normal odors; and the other in which odors are imaginary (cacosmia). Both conditions occur in certain nervous affections, among the insane, in epilepsy (aura), and occasionally in disease of the olfactory nerve or its terminal filaments.

V. Taste

The sense of taste may be impaired, perverted or lost This may be due to local conditions of the mouth or nose and to nerve paralysis.

Local Conditions: The taste may be lost or perverted in the various types of stomatitis and glossitis, in nasal obstruction, in diseases of the gastrointestinal tract, and in febrile diseases associated with a heavily coated or exceedingly dry tongue. The sense of taste may be impaired from taking certain articles of food or drugs.

Nerve Paralysis: In peripheral facial and in trigeminal nerve palsy the sense of taste may be lost on the anterior two-thirds of the tongue on the paralyzed side to sweets, bitters, salty or sour articles.

In some of the neuroses and in digestive disorders due to gastric or hepatic conditions certain tastes may be persistent, irrespective of the kind of food taken. Some patients may complain of a persistent bitter taste, others of a constant sweet taste, or there may be a sour, salty, or metallic taste felt on the tongue, the lips or within the mouth generally.

CHAPTER IV

Pain and Tenderness

I. The Nature of Pain

Pain is a protective function, part of a defensive mechanism appraising the individual of injury to vital tissue.

Painful sensations are transmitted through the sensory nerves of a part to the pain center and redirected, in most cases, to the site of the injury. When the nerve is anesthetized or blocked, or the center is destroyed, pain is not perceivable. Pain is one of the commonest symptoms for which the physician is consulted. It is usually the most important of all symptoms to the sufferer. The degree and kind of pain cannot, as a rule, be judged by the examiner, he therefore must rely to a great extent on the patient's description of his sensations and on his physical and mental reaction. The hyperesthetic or pain-sensitive individual will react intensely to moderate pain, while the stoic may effectively mask a severe degree of pain. The description of the type of pain often depends upon the individual's descriptive ability. Therefore it is necessary to evaluate the person's sensitivity and to watch closely his mannerisms and his actions when describing the pain he has suffered or is suffering at the time of the examination. The sensitivity of an individual may be roughly gauged, as shown by E. Libman, by his responses to pressure over a bony prominence as, for example, over the ulnar prominence at the wrist or over the petrous portion of the temporal bone.

Pain over the entire body is uncommon; it is nearly always localized either over a limited, or an extensive area. Pain *per se* is not a disease, but a symptom of injured tissue. While it is often of great importance to relieve the pain, it is of still greater importance to determine the reason for it so that adequate treatment may be instituted to prevent or correct the defect causing the condition which is responsible for the pain. Pain may be felt at the site of injury or it may be felt at a distance from the injured area (referred pain). Pain may be continuous, intermittent, or remittent. It may be colicky, sharp, stabbing, lancinating, or dull and aching; it may also be throbbing, expanding or compressing. Pain may be constant, or it may be provocative, that is, brought out by moving or by manipulating the affected part; and it may be superficial, deep seated or migrating. Pain of equal intensity cannot, as a rule, be felt in several places at the same time.

Tenderness is a painful condition brought about by pressure; it may be superficial where the mere touching of the skin causes pain, or deep seated as in inflammations of deep-seated organs or bone. Deep-seated tenderness is usually associated with rigidity of the overlying muscles.

II. Physical Signs of Pain

While pain is only a symptom perceived by the patient, there are nevertheless certain signs by which the examiner may in a general way judge the intensity of the patient's suffering. From the standpoint of physical signs, pain may be subjective or objective.

Subjective Pain: This has no apparent physical basis for its existence; it may be found among the highly imaginative neurotics where mild sensations are translated into pain sense, particularly when they are or recently were in contact with a person who had severe pain of a serious nature, as coronary occlusion or perforated ulcer. It also occurs in hysteria. Pain in these individuals is not constant nor is it confined persistently to one location, and their physical reactions, such as moaning, complaining, wincing and assumed postures are entirely out of proportion to the reactions usually seen in nonneurotics who may have an injury causing that type of pain. It must be borne in mind, however, that a neurotic and hysterical person may actually suffer a physical injury or disease which may cause much pain, and because his reactions are more intense than is the general rule, he should not be summarily dismissed as a "neuro" suffering from subjective pain. Many a so-called "neuro" has come to an untimely grave because it was believed that he "cries wolf too often." The pains of hysteria and hypochondria may have central nervous system origin even though a physical cause be absent. Subjective pain is as real to the neurotic as are dreams to the sleeper. During a dream an individual may experience many and varied sensations which he believes are real and thus may suffer untold agony or great pleasure; so the neurotic, during his painful episodes, suffers as much and as keenly as if his pains had a definite physical basis. However, his pains may diminish in intensity or even disappear when his attention is diverted from them, and they may be aggravated by suggestion. Nervousness, fright, anxiety, expectations, anger, and disappointment intensify painful impressions in neurotic individuals.

Objective Pain: This is excited by some external or internal irritant, by inflammation, or by injury to nerves, organs or other tissues which interfere with the function, nutrition or circulation of the affected part Such pain is usually traceable to a definite pathologic process.

III. Type of Pain

The type of pain varies with the tissues affected.

Acute Pain: *Sharp, lancinating,* or *stabbing* pain is usually associated with acute inflammation of a nerve, nerve endings or of the serous membranes covering a viscus as in pleurisy, pericarditis, peritonitis, neuralgia, neuritis, and posterior spinal nerve root pains. Pain of similar character and intensity is often found in acute arthritis, thoracic aneurysm, tumor of the spinal cord, tabes dorsalis, and herpes zoster

Pressing, Aching, Agonizing Pain: *In the chest* this may be due to coronary thrombosis, angina pectoris, aortic aneurysm, mediastinitis, and, in a milder form, it may occur in asthma and tracheobronchitis; it may also be due to referred pain from a diseased gallbladder, an intestinal obstruction, a diaphragmatic hernia, pancreatitis and a perforated ulcer

of the stomach. *Aching generalized pains* usually precede or are ushered in with some of the infectious diseases as influenza, dengue, smallpox, rheumatic fever. *Locally*, aching pains are also found in myalgia, lumbago and various types of headache.

Throbbing Pain: This type is often associated with phlegmonous inflammation and suppuration, and is also found in headache and in dental caries.

Colicky, Griping Pains or Cramps: These types of pain are found in various intestinal disorders associated with flatulence and contractions of the intestines such as are found in cholera morbus, Asiatic cholera, and after ingestion of irritating poisons, indigestible food, or strong cathartics; also in biliary colic, renal colic, Dietl's crises, pancreatitis, intestinal obstruction, strangulation, appendicitis, colitis, ruptured tubal pregnancy, torsion of an ovary, dysmenorrhea, orchitis, etc. *Muscle cramps* may be due to strychnia poisoning, intermittent claudication, tetanus, tetany, muscle strain, muscle ischemia; and are also seen as the result of certain occupations, such as writer's cramps, piano or violin player's cramps, chauffeur's cramps, telegrapher's cramps, etc.

Causalgia: *Burning pains* are found in sunburn or other heat burns, in certain superficial skin lesions, in circumscribed neuralgias, and in herpes zoster.

Grinding or Gnawing Pain: This type is quite characteristic of diseases of bone and periosteum. It is also at times encountered in aneurysm of the abdominal aorta and in carcinoma of the viscera and of the breast.

Dull Pain: It occurs in inflammation of the mucous membranes and the viscera; it also occurs in chronic inflammatory conditions.

IV. Location of Pain

Pain may be felt only over the site of the diseased organ or tissue, or it may be felt over a distant part of the body to which it may be referred by the diseased part. In coronary occlusion the pain is felt behind the sternum and is referred to the left and, at times, to the right arm, the shoulders or neck; in biliary colic, pain is felt over the gallbladder and is referred to the right shoulder; in renal colic, the pain is felt in the kidney region and is referred to the urinary bladder or lower.

Referred Pain: In some instances, pain is felt not over the diseased area or organ but at some distance from it. This is particularly true in diseases of the viscera. The reason for the transferred sensitivity is explained by Head as follows:

Head's Law: "When a painful stimulus is applied to a part of low sensibility in close central connection with a part of much greater sensibility, the pain produced is felt in the part of higher sensibility rather than in the part of lower sensibility to which the stimulus was actually applied."

V. Regional Pain

A. Headache (Cephalalgia)

Headache is one of the commonest of complaints; it may occur in otherwise perfectly normal individuals as the result of some trifling or functional disorder; or it may be a sign of a serious and severe malady. Headache may be due to local conditions as disease of the men-

inges or brain; or it may be referred from some distant diseased organ. Toxemias, fever, disturbed circulation and exhaustion may cause headache, as will also local disease of the cranial bones and their coverings. Headache may be constant with periods of remission and exacerbation of the severity of the pain, and it may be periodic or transient. The character of the pain, its location and the accompanying symptoms and signs must be considered before a diagnosis of its cause can be reached

Headache Due to Intracranial Lesions[1]

Brain Tumor: Here the headache is constant Occasionally the pain and some tenderness overlie the location of the growth Rapidly growing tumors cause more intense pain than slowly growing tumors. The pain is less intense in gliomata than in other cerebral neoplasms The character of the pain varies; it may be dull and boring, or lancinating and agonizing; it is, as a rule, continuous with periods of exacerbation and is usually most severe at night The pain may be localized or diffused Other diagnostic aids are eye examination for signs of choked discs, papilledema and hemianopsia; brain localization phenomena; the degree of intraspinal pressure; x-ray examinations and ventriculographic studies. Most intracranial space-taking lesions present localizing symptoms and such general symptoms as headache, vomiting, mental drowsiness, dizziness, alteration of pulse rate, respiratory rate, blood pressure and not infrequently convulsions

Cerebral Abscess: The headache is constant and severe and is usually localized over the affected area. Fever, vomiting, vertigo, mental dullness, irritability and general weakness usually accompany the localized pain and the general headache.

Aneurysm: Aneurysm of one of the *intracranial vessels* usually causes expansile or throbbing headache which is felt over the entire head or at the occiput The pain may be continuous or paroxysmal; it is usually aggravated on physical or mental exertion Accompanying signs may be intracerebral pressure symptoms, diabetes insipidus and general irritability. Caries of the bones of the skull and affections of the scalp due to aneurysm of an intracranial vessel may present, in addition to the more or less boring and lancinating headache, areas of local tenderness and pulsation.

Cerebral Concussion: This gives rise to severe protracted headache which may be localized or diffused. It may be felt over the site of the injury or on the opposite side of the head. It is usually associated with superficial tenderness, and at times with other evidence of injury and with vertigo, lassitude and mental confusion.

Cerebral Hemorrhage: When not sufficiently extensive to cause unconsciousness, it will cause severe boring pain over the frontal or occipital regions, and may be accompanied by irregular pupils, hemiplegia, bulbar compression signs or other intracranial pressure symptoms, depending upon the site and magnitude of the hemorrhage.

Meningitis: Headache is present in all types of meningeal irritation. The headache of intracranial lesions is largely due to meningeal involvement, since the brain, while the perceptor of pain sense elsewhere in the body, is itself, when traumatized, insensible to pain. The pain in meningitis is intense and agonizing

[1] SEE: *Pituitary Headache*, p 773, and *Lesions of the Brain*, p 866

It may be localized in local inflammation and is generalized in the various types of meningitis. The associated symptoms are fever, nuchal rigidity, increased intraspinal pressure, changes in cerebral fluid composition, and such signs as Kernig's, Brudzinski's, Babinski's, Hoffman's, etc

Infections: Most of the infectious fevers are ushered in with headache, in some the headache is acute and agonizing and is associated with generalized pain The headache does not, as a rule, persist throughout the entire course of the disease.

Sinusitis: Particularly when frontal and ethmoidal, sinusitis causes severe excruciating pain in the frontal region.

Toxemia (*Acute or chronic*): If caused by drugs, endotoxins, exotoxins, or by gastrointestinal disturbances, such as constipation, toxemia may cause dull generalized headache or acute pain in the temporal regions or over the vertex.

Reflex Headache: This may be dull and protracted or acute, intense and of short duration or it may be paroxysmal. The headache may affect any portion of the head; it may be of varying intensity or type and may resemble organic disease. Among the conditions causing reflex headache may be mentioned eyestrain, certain eye diseases, tooth affections, diseases of the ear, gonad disturbances, toxemia, renal disease, uremia, overwork, exhaustion, lack of sleep, emotional states, arteriosclerosis, hypertension, hypotension, hyperemia, cardiac decompensation, anemia, spinal puncture, exophthalmic goiter, rheumatic affections, myalgia of the scalp, neuralgia, cervical adenitis, Ménière's disease, the various neuroses and psychoses, vasomotor disturbances and diseases of the cranial bones.

Headache is also common in sunstroke, heat exhaustion, insulin shock, trigeminal neuralgia, etc. Occasionally, headaches of various types and severity may occur without any obvious cause. Syphilis and histominic cephalgia should not be overlooked in obscure headache.

Migraine

Migraine is a paroxysmal familial special type of headache. It is characterized by hemicrania (at times it may be bilateral) associated with visual, gastric and nervous phenomena suggesting brain cortex involvement.

Symptoms: In the great majority of cases there is a history of one or more members of the family who are or have been subject to migraine, showed allergic sensitivity, or suffered from diabetes, epilepsy or some endocrinopathy The attacks of pain may be preceded by a prodromal period which may last from 8 to 12 hours. The prodromal symptoms vary in different individuals. There may be depression or hyperactivity, somnolence or insomnia, excessive appetite or complete anorexia with varying digestive disturbances. Immediately before the attack there may be an aura, though that is not constant nor is it always of the same character. The aura may consist of vertigo, photophobia, lacrimation, scotomata, blurred vision, olfactory changes, coldness, sweating, paresthesia of the extremities, and other sensory and motor changes or mental confusion

The attack usually commences on waking in the morning, though it may come on at any time. The patient develops a feeling of seasickness, vertigo, intense pain in some part of the head (usually over one eye or hemicrania), vomiting and visual disturbance, and often sensory, motor and psychic disturb-

ances The headache is cumulative and expansile in character; it may be unilateral, localized in a temple or in an eyeball or upon the forehead. It is sharp and boring, may spread over the entire head and may involve the neck and arm There may be soreness of the eyeballs and hyperesthesia of the scalp. During the attack the patient is pale, prostrated, incapable of mental or physical effort, and usually assumes a definite posture in bed from which he would not be disturbed Light, noise, solicitude and other disturbances as well as movement aggravate the condition. The attack may last from 3 to 24 hours or longer.

Etiology: Migraine usually occurs in adolescents and young adults, and generally disappears after the menopausal age. Heredity plays a part since the syndrome is familial The actual cause of migraine is not known; there are several theories but no facts. Allergy, duodenal stasis, endocrine disturbance, reflex causes (eyestrain, disgestive disturbance, etc.), toxic causes from the colon or elsewhere, vasomotor disturbance or cortical disturbance are among the supposed etiologic factors

B. Pain in the Eyes[1]

Pain in the eyeballs may range from a smarting, burning or "sand in the eyes" sensation to acute, excruciating pain. Pain in the eyes may be due to eyestrain, general fatigue, conjunctivitis, foreign bodies in the eye and traumatism Conjunctivitis may cause either mild or very intense pain depending upon the extent of the inflammation. The pain in corneal ulcer depends upon the location of the ulcer, its depth and the amount of inflammatory reaction. Occasionally a corneal ulcer may be painless In keratitis the pain is usually severe and is accompanied by photophobia, blepharospasm and lacrimation. In iritis the pain is often severe and is felt as if originating in the eyeball It is referred around the orbit, in the temple and forehead; the pain is worse at night. Acute glaucoma causes severe excruciating pain in the eyeball associated with severe headache and is often accompanied by nausea, vomiting, general depression, and a rise in temperature. In panophthalmitis and suppurative iridochoroiditis the pain in the affected eye is agonizing and is accompanied by marked conjunctivitis, haziness of the cornea and swelling of the lids. In acute retrobulbar neuritis the pain is felt in the affected orbit, it is aggravated on pressing the eyeball or by movement of the eye, and there is severe headache on the affected side In sphenoidal sinusitis there is deep-seated pain in the eyes, and headache. Migraine may, in addition to the severe headache, also cause pain first in one eye and later in both eyes. The pain in the eyes is often described as though the eyes were either being gouged out of their sockets or forcibly pressed inwards. In some of the acute fevers pain in the eyeballs is a frequent complaint. This is found particularly in influenza, typhoid fever, typhus fever, smallpox, measles, malaria, coryza and other infections.

[1] See also: pp 171-182

C. Glossalgia

Pain in the tongue may be due to lesions upon the tongue, to gastrointestinal diseases and to certain anemias

Lesions upon the tongue causing pain are:

(*a*) **Ulcerations:** They may be due to trauma, such as bites, mechanical injury, sharp projections from a defective tooth or from artificial denture.

It may be localized in local inflammation and is generalized in the various types of meningitis The associated symptoms are fever, nuchal rigidity, increased intraspinal pressure, changes in cerebral fluid composition, and such signs as Kernig's, Brudzinski's, Babinski's, Hoffman's, etc.

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The pain may be traced from the spine following the course of the affected intercostales to the sternum A tender area is usually located at points where the terminal filaments reach the surface or where the affected nerve emerges from the spine.

(*c*) **Disease or Injury:** Trauma affecting the ribs, sternum or spine as in tuberculosis, osteomyelitis, malignancy, and in erosions by aneurysm or other disease processes of any of these structures causes sharp pain on motion.

(*d*) **Arthritis, Arthalgia or Synovitis:** When these conditions affect the spinal or sternal rib articulations the pain is usually aggravated on motion or breathing

(*e*) **Diaphragmatic Pleurisy and Subdiaphragmatic Abscess:** In these conditions, the pain may be sharp or dull and is aggravated by deep breathing, by abdominal distention and by straining.

(*f*) **Pleurisy:** *Acute pleurisy* causes localized sharp stitchlike pain on breathing It may be of rheumatic, tuberculous, influenzal, streptococcic or of other bacterial origin It is often seen at the onset of lobar pneumonia, in endothelioma; or it may be traumatic in origin. *Chronic pleuritis* may be associated with neoplasm, tuberculosis, pulmonary suppurations, pericarditis, aneurysm, disease of the ribs and spine, and empyema. Here the pain is sharp but not as sharp as in acute pleuritis. The pain is aggravated by deep breathing, coughing, sneezing, yawning, laughing, singing, and loud crying or talking. Immobilizing the chest, the absorption of the sticky exudate, or the formation of effusion eases or stops the pain.

(*g*) **Disease of the Lungs:** This seldom causes pain unless the pleura is involved Spontaneous pneumothorax will cause such acute sharp pain that it immobilizes the chest. Accompanying the pain there is a sense of expansion which at times is expressed by some patients as crushing or compressing. The pain may be referred to the diaphragm or to the neck and the axillary region. The physical signs of pneumothorax will establish the diagnosis

(*h*) **Diseases of the Heart and Aorta:** Pericarditis if dry, whether tuberculous or rheumatic, is usually accompanied by pain, and a friction rub is, as a rule, audible over some part of the precordium Aortitis and aortic regurgitation occasionally cause pain in the chest which is aggravated by physical exertion. Coronary occlusion causes severe, excruciating, pressing pain in the sternal and occasionally in the epigastric region; it is referred to the ulnar distribution of the left upper extremity, occasionally to the shoulder and at times to the right arm Angina pectoris, whether due to coronary sclerosis, aortitis, or other causes, produces pain similar to that felt in coronary occlusion. The duration is shorter and the pain is of lesser severity and is frequently brought on by exertion Occasionally such pain develops while the patient is in bed and is relieved on assuming an erect posture.

(*i*) **Mediastinal Tumors:** These, when they particularly crowd the aorta and the sensory nerves, may cause substernal or intercostal pain.

(*j*) **Referred Pain:** Pain in the chest is occasionally referred from disease originating below the diaphragm This is seen in subdiaphragmatic abscess, liver abscess, cholecystitis, cholelithiasis, retroperitoneal malignancy, peritonitis, particularly affecting the lesser peritoneal cavity, pancreatitis, splenic infarcts, nephrolithiasis, hydronephrosis, suprarenal

(*b*) **Fissures:** They may be caused by gastrointestinal disease, syphilis, local or general irritation from smoking, hot food or other irritating substances. They may also be found in mouth breathers, in generalized dryness of the tongue, in avitaminosis and occasionally the cause is not discoverable. The pain is sharp and is aggravated by spicy food.

(*c*) **Acute Glossitis:** This condition occurs in vitamin deficiency, anemia and, idiopathically, chiefly in women of neurotic tendencies. The lesions occur as isolated white patches with small erosions having ragged edges; the pain is of a burning character.

(*d*) **Chronic Superficial Glossitis** (*Moeller's glossitis*): It occurs as red or erythematous areas upon the dorsum, but chiefly at the margin and top The pain is peppery or burning; or the tongue feels as if it were scalded. It is aggravated by talking and eating, particularly if the food is spicy. Soon after eating, the pain subsides but returns several hours later. This occurs more often in women than in men.

(*e*) **Abscess of the Tongue:** It may be primary or secondary to mouth infection; the pain is more intense on talking and chewing.

(*f*) **Geographic Tongue:** It may cause burning pain when denuded surfaces or fissures develop among the peculiar patterns.

(*g*) **Tuberculosis,** (*h*) **Syphilis,** and (*i*) **Carcinoma:** They do not cause pain until ulcerations develop or when the lesions interfere with lingual mobility or when there is glandular swelling.

(*j*) **Eczema of the Tongue:** This occurs in patches; the pain is a burning sensation aggravated by irritating foods.

(*k*) **Pyorrhea Alveolaris:** Stomatitis, any type of xerostomia, or other mouth infections may cause pain and burning of the tongue.

(*l*) **Glossodynia:** Painful tongue without local lesions is found in the various neuroses, trigeminal neuralgia, in tabes dorsalis and occasionally in otherwise normal persons.

Pain in the tongue occurs in various deficiency diseases, such as pellagra, scurvy, chronic steatorrhea, and sprue. It also occurs in chronic liver and gall-bladder affections, in mucous colitis, regional ileitis and occasionally in malignancy of the digestive system Glossitis or glossodynia is frequently found in pernicious anemia, chlorosis and also in the various secondary anemias, particularly of the macrocytic hyperchromic and the microcytic hypochromic types

D. Pain in the Chest

Pain in the chest is felt when the chest wall or its inner lining is irritated or inflamed. This includes the skin, the costal and intracostal muscles, the periosteum, the pleurae, the pericardium and the spine; also certain affections of the aorta, coronary vessels and mediastinum The heart and lungs, when diseased, cause pain only when their serous covering becomes inflamed or is injured, or when there is interference with their blood supply.

Pain in the chest may be due to a variety of causes.

(*a*) **Various conditions** that affect the thoracic wall may be inflammatory lesions, tumors, skin lesions, muscle injury, neuralgia, neuritis, and herpes zoster. The pain is usually superficial, the area affected is tender to touch and the pain may be aggravated on motion.

(*b*) **Intercostal Neuralgia:** This is characterized by sharp pain, aggravated on breathing and relieved by pressure

accompanied by boardlike rigidity and tenderness over the upper abdomen.

Pylorospasm: This usually comes on as a cramplike epigastric pain with a sense of distention or expansion in the upper abdomen two or three hours after meals and may last from five minutes to one-half hour or longer.

Acute Hemorrhagic Pancreatitis: This is manifested by sudden severe colicky pain in the epigastrium, upper abdomen, and occasionally over the entire abdomen; and is accompanied by copious bile-stained vomiting, abdominal distention, a sense of resistance in the upper abdomen, and by shock or collapse, a subnormal temperature and leukocytosis.

Chronic Pancreatitis: This may at times cause epigastric pain, nausea, vomiting and jaundice; the pain is usually referred to the left hypochondrium and downward.

Cholelithiasis and Cholecystitis: They may often cause pain in the epigastrium The pain may be referred to the upper chest, the back or to the right shoulder posteriorly. Jaundice and clay-colored stools will occur when there is obstruction of the common bile duct Hepatic enlargement due to abscess, cyst, carcinoma, acute congestion, syphilis and cirrhosis may also cause pain in this region

Nephrolithiasis: It may occasionally cause epigastric pain which is acute and colicky, but is generally referred downward toward the urinary bladder.

Abdominal Angina: Angina pectoris may at times simulate biliary colic, "acute indigestion", pancreatitis, or other acute abdominal catastrophes. The onset of the pain is sudden and severe and may be referred backward to the spine or upward beneath the sternum. It may come on after exertion, emotional stress or after a heavy meal, and may last from a few minutes to several hours. There is usually a sense of impending death and copious perspiration. The pain is viselike and is associated with precordial tenderness. Belching or vomiting often terminates the attack.

Tabes Dorsalis (Locomotor ataxia): The pain is sudden, acute and colicky. It is encircling or beltlike in distribution. The pain is not dependent on the gastric or intestinal contents. Vomiting, pallor, sweating and a small and feeble pulse occur during the attack. The presence of signs of cerebrospinal syphilis, in the absence of other pathology, suggests tabetic crises.

Retroperitoneal Malignancy: This will often resemble acute gastric, pancreatic or biliary tract disease. The failure to find evidence of disease in these structures by x-ray examination and by laboratory tests will exclude disease of these organs. The pain in retroperitoneal malignancy is sharp, lancinating and may be referred to various parts of the abdomen and is not related to food or to bowel action. It is not relieved by alkalies or antispasmodics.

Abdominal Aneurysm: It causes expansile, localized abdominal pulsation, and at times a *bruit* may be audible. The pain is not related to food. A positive serologic test for syphilis, in the absence of pathologic findings, in the gastrointestinal tract or in the spinal column suggests the possibility of aneurysm.

Omental Hernia: It may be inferred from the acute pain, shock, generalized abdominal distention, silent abdomen and other signs of intestinal obstruction

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tumors, disease of the spine, gastric or duodenal ulcers, intestinal obstruction, mesenteric thrombosis, appendicitis, orchitis, tuboovarian disease, and in inflammatory diseases of the abdominal wall. In the presence of severe or persistent chest pains, when thoracic pathology is absent, a thorough abdominal examination is necessary.

E. Pain in the Abdomen

Pain in the abdomen may be generalized over the entire abdomen or it may be localized in any part of the abdomen. The pain may be acute, colicky or it may be dull; and there may be associated with it tenderness on pressure, general distention and local or general muscle spasm or rigidity. The pain may be due to inflammation or injury of the abdominal wall or to disease of the abdominal viscera Abdominal pain due to visceral disease occurs as the result of inflammation of the peritoneum, inflammation or injury to the serous covering of the various organs, interference with their blood supply, hypertraction upon the tissues carrying their nerve and blood supply, and inflammation or injury to the sensory nerves.

General Abdominal Pain: This is a common symptom in acute generalized peritonitis, acute hemorrhagic pancreatitis, ruptured gastric ulcer, mesenteric thrombosis, acute gastroenteritis, acute enterocolitis, idiopathic steatorrhea, acute intestinal obstruction (due to adhesive bonds, strangulated hernia, intussusception, volvulus, paralytic ileus), chronic intestinal obstruction, tumors of the large or small intestines, food poisoning, ulcerative colitis, mucous colitis, spastic colitis, amebic dysentery, bacillary dysentery, lead, arsenic, mercury and other metallic poisonings, tabes dorsalis, crises of Addison's disease and of exophthalmic goiter, Asiatic cholera, achylia gastrica, abdominal aneurysm, atheroma of the abdominal aorta, Hirschsprung's disease, rupture of an abdominal viscus, tuberculous peritonitis, torsion of an ovarian cyst, abdominal neoplasm, disease of the dorsal or lumbar spine, and disseminated sclerosis Occasionally, general abdominal pain occurs at the onset of acute appendicitis, and in regional ileitis, retroperitoneal malignancy, chronic constipation, allergic dyspepsia, intestinal worms, orchitis, morphinism, purpura hemorrhagica and other blood diseases, periarteritis nodosa and intestinal neurosis. The pain may also be referred to the abdomen from the chest as in pneumonia, pleurisy, angina pectoris and coronary sclerosis.

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cause acute pain with shock. Splenic infarcts cause *sudden violent pain in the* splenic region and are accompanied by splenic enlargement Splenomegaly associated with abscess, tuberculosis, amyloid disease and acute congestions will cause dull pain and tenderness Rupture of the spleen will cause acute pain followed by shock and signs of internal hemorrhage. Obstruction of the bowel, fecal impaction, carcinoma of the splenic flexure or of the descending colon, mucous colitis, spastic colon, and diverticulitis will cause colicky pain in proportion to the extent of the abdominal distension. Disease of the pancreas, hyperdistension of the stomach, diaphragmatic hernia and occasionally cholecystitis and cholelithiasis may cause referred pain to the left side of the abdomen. Referred pain in the left hypochondrium may have etiologic factors similar to those that cause pain in the right hypochondrium.

Pain in the Right Loin: This type of pains may be caused by nephrolithiasis; the passage of the stone through the right ureter causes pain in the loin which may be referred to the hypochondrium and downward toward the genitalia (penis, testes or vulva) and occasionally to the perineum and the inner side of the thigh. The pain is colicky in character and is often intermittent. *Torsion of the ureter caused by a floating* kidney or stricture of the ureter will cause the same type of pain Pain in the right loin may also be caused by hydronephrosis, pyonephrosis, pyelitis, nephritis, large tuberculous kidney, polycystic kidney, various tumors, cysts and abscesses of the kidney and of the adrenals, aneurysm of the renal artery, lumbosacral sprain, disease of the lumbar vertebrae, inflammatory nucleus pulposus, spinal tumors, *orchitis, irritation of the 12th dorsal and* 1st lumbar nerves, fracture of the 11th *or 12th ribs, foot strain, and by fibrositis* affecting the lower intercostales and the muscles and nerves in that region. In addition to pain in the right loin and occasionally in the hypochondrium, there are other symptoms and physical signs by which many of these conditions can be identified.

Pain in the Left Loin: This may be caused by left-sided renal calculus, left-sided hydronephrosis, pyonephrosis, pyelitis, kinks and other obstructions in the left ureter, tumors, abscesses, cysts, infarcts and inflammatory disease of the left kidney and by tumors, cysts and abscesses of the left adrenal.

Pain in the Iliac and Inguinal Regions: ***Acute Appendicitis:*** The pain is localized in the right iliac region near McBurney's point. When the appendix is retrocecal, the pain is felt low down *in the inguinal region and when* the appendix points upward, the pain is referred to the upper right abdomen. The pain is colicky or it may be constant and severe and is aggravated by palpation. In addition to the pain there is rigidity of the lower part of the right rectus muscle. There is usually a moderate leukocytosis and some rise in temperature. Rupture of the appendix causes a temporary lull of pain and of rigidity which is later fol*lowed by signs of peritonitis.*

Acute Salpingitis: This causes right or left-sided severe lancinating paroxysmal pain; it is not as strictly circumscribed as appendiceal pain and it radiates to the thighs

Ruptured Ectopic Gestation: The pain is sudden and severe, often accompanied by shock or collapse, particularly so if the accompanying hemorrhage is copious. The pain is bearing down in character and radiates to the umbilicus.

partially strangulated, it will cause severe epigastric pain, referred to the left chest and will be accompanied by signs of obstruction, i. e., vomiting, distention, etc.

Periarteritis Nodosa: This often causes severe epigastric pain, or severe pain in the upper right or upper left abdomen It is accompanied by rigidity, irregular temperature, leukocytosis, hypertension and signs of systemic disease

Pain in the Right Hypochondrium: This may be caused by disease of the structures which are situated in that region or it may be referred from adjacent or remote structures The commoner causes for pain in the right hypochondrium are·

Cholelithiasis: A gallstone in its attempt to pass through a channel whose lumen is too small to permit its free passage will cause obstruction with consequent dilatation above the point of obstruction This produces severe colicky pain A similar type of pain may be caused by *inflammation of the gallducts* and *pressure upon the gallducts or gallbladder*. The pain is colicky, usually intermittent, and it is felt in the region of the gallbladder and the epigastrium It is as a rule referred to the back and up to the right shoulder When obstruction is complete, jaundice develops. A stone passing through the pancreatic duct or one lodged in the common or the cystic duct will cause a similar type of pain in the right hypochondrium and epigastrium.

Cholecystitis without Calculi: This condition may cause the same type of pain as calculous cholecystitis, particularly when the bile duct becomes occluded because of inflammation and the gallbladder becomes hyperdistended This type of pain also occurs in acute cholecystitis and in empyema of the gallbladder.

Subphrenic Abscess: The abscess causes constant pain which is aggravated on breathing and by pressure. The pain may be referred to the clavicular region by the phrenic nerve, to the intercostal spaces by the intercostal nerves, or towards the lower abdomen and loin because of pressure upon the liver, adrenal or kidney.

Subdiaphragmatic Pleurisy: It causes sharp stitchlike pain during breathing. When the breath is held, the pain ceases Pressure against the lower costales eases the pain.

Diseases of the Liver: Such diseases as carcinoma, cysts, abscess, biliary cirrhosis, gumma and acute passive congestion usually cause pain and tenderness in the liver region. The pain may be constant and draggy and is aggravated by palpation

Various Other Causes: Pain in the right hypochondrium may also be caused by right-sided spontaneous pneumothorax, pneumoperitoneum, right-sided basal pleurisy, pneumonia, carcinoma, empyema or other diseases of the lung and pleura Other causes of pain in this region may be herpes zoster (before the appearance of the rash), disease of the spine, carcinoma of the hepatic flexure or ascending colon and acute appendicitis when the appendix is pointing upward.

Pain in the Left Hypochondrium: It may be caused by left-sided spontaneous pneumothorax, where the pain is sudden and severe and is accompanied by immobility of the chest and other signs of pneumothorax Left-sided diaphragmatic pleurisy causes increased pain on breathing and is relieved by pressure. Diaphragmatic hernia will

cause acute pain with shock. Splenic infarcts cause sudden violent pain in the splenic region and are accompanied by splenic enlargement Splenomegaly associated with abscess, tuberculosis, amyloid disease and acute congestions will cause dull pain and tenderness. Rupture of the spleen will cause acute pain followed by shock and signs of internal hemorrhage Obstruction of the bowel, fecal impaction, carcinoma of the splenic flexure or of the descending colon, mucous colitis, spastic colon, and diverticulitis will cause colicky pain in proportion to the extent of the abdominal distension Disease of the pancreas, hyperdistension of the stomach, diaphragmatic hernia and occasionally cholecystitis and cholelithiasis may cause referred pain to the left side of the abdomen. Referred pain in the left hypochondrium may have etiologic factors similar to those that cause pain in the right hypochondrium.

Pain in the Right Loin: This type of pains may be caused by nephrolithiasis; the passage of the stone through the right ureter causes pain in the loin which may be referred to the hypochondrium and downward toward the genitalia (penis, testes or vulva) and occasionally to the perineum and the inner side of the thigh The pain is colicky in character and is often intermittent Torsion of the ureter caused by a floating kidney or stricture of the ureter will cause the same type of pain Pain in the right loin may also be caused by hydronephrosis, pyonephrosis, pyelitis, nephritis, large tuberculous kidney, polycystic kidney, various tumors, cysts and abscesses of the kidney and of the adrenals, aneurysm of the renal artery, lumbosacral sprain, disease of the lumbar vertebrae, inflammatory nucleus pulposus, spinal tumors, orchitis, irritation of the 12th dorsal and 1st lumbar nerves, fracture of the 11th or 12th ribs, foot strain, and by fibrositis affecting the lower intercostales and the muscles and nerves in that region. In addition to pain in the right loin and occasionally in the hypochondrium, there are other symptoms and physical signs by which many of these conditions can be identified.

Pain in the Left Loin: This may be caused by left-sided renal calculus, left-sided hydronephrosis, pyonephrosis, pyelitis, kinks and other obstructions in the left ureter, tumors, abscesses, cysts, infarcts and inflammatory disease of the left kidney and by tumors, cysts and abscesses of the left adrenal.

Pain in the Iliac and Inguinal Regions: ***Acute Appendicitis:*** The pain is localized in the right iliac region near McBurney's point. When the appendix is retrocecal, the pain is felt low down in the inguinal region and when the appendix points upward, the pain is referred to the upper right abdomen. The pain is colicky or it may be constant and severe and is aggravated by palpation. In addition to the pain there is rigidity of the lower part of the right rectus muscle. There is usually a moderate leukocytosis and some rise in temperature. Rupture of the appendix causes a temporary lull of pain and of rigidity which is later followed by signs of peritonitis.

Acute Salpingitis: This causes right or left-sided severe lancinating paroxysmal pain; it is not as strictly circumscribed as appendiceal pain and it radiates to the thighs

Ruptured Ectopic Gestation: The pain is sudden and severe, often accompanied by shock or collapse, particularly so if the accompanying hemorrhage is copious. The pain is bearing down in character and radiates to the umbilicus

partially strangulated, it will cause severe epigastric pain, referred to the left chest and will be accompanied by signs of obstruction, i. e., vomiting, distention, etc.

Periarteritis Nodosa: This often causes severe epigastric pain, or severe pain in the upper right or upper left abdomen It is accompanied by rigidity, irregular temperature, leukocytosis, hypertension and signs of systemic disease

Pain in the Right Hypochondrium: This may be caused by disease of the structures which are situated in that region or it may be referred from adjacent or remote structures. The commoner causes for pain in the right hypochondrium are·

Cholelithiasis: A gallstone in its attempt to pass through a channel whose lumen is too small to permit its free passage will cause obstruction with consequent dilatation above the point of obstruction This produces severe colicky pain A similar type of pain may be caused by *inflammation of the gallducts* and *pressure upon the gallducts or gallbladder* The pain is colicky, usually intermittent, and it is felt in the region of the gallbladder and the epigastrium It is as a rule referred to the back and up to the right shoulder When obstruction is complete, jaundice develops A stone passing through the pancreatic duct or one lodged in the common or the cystic duct will cause a similar type of pain in the right hypochondrium and epigastrium.

Cholecystitis without Calculi: This condition may cause the same type of pain as calculous cholecystitis, particularly when the bile duct becomes occluded because of inflammation and the gallbladder becomes hyperdistended This type of pain also occurs in acute cholecystitis and in empyema of the gallbladder.

Subphrenic Abscess: The abscess causes constant pain which is aggravated on breathing and by pressure. The pain may be referred to the clavicular region by the phrenic nerve, to the intercostal spaces by the intercostal nerves, or towards the lower abdomen and loin because of pressure upon the liver, adrenal or kidney

Subdiaphragmatic Pleurisy: It causes sharp stitchlike pain during breathing When the breath is held, the pain ceases. Pressure against the lower costales eases the pain

Diseases of the Liver: Such diseases as carcinoma, cysts, abscess, biliary cirrhosis, gumma and acute passive congestion usually cause pain and tenderness in the liver region The pain may be constant and draggy and is aggravated by palpation.

Various Other Causes: Pain in the right hypochondrium may also be caused by right-sided spontaneous pneumothorax, pneumoperitoneum, right-sided basal pleurisy, pneumonia, carcinoma, empyema or other diseases of the lung and pleura Other causes of pain in this region may be herpes zoster (before the appearance of the rash), disease of the spine, carcinoma of the hepatic flexure or ascending colon and acute appendicitis when the appendix is pointing upward

Pain in the Left Hypochondrium: It may be caused by left-sided spontaneous pneumothorax, where the pain is sudden and severe and is accompanied by immobility of the chest and other signs of pneumothorax Left-sided diaphragmatic pleurisy causes increased pain on breathing and is relieved by pressure. Diaphragmatic hernia will

disease of the spine, bladder, uterus, prostate, hip joint, rectum and lower bowel. Diseases of the abdominal arteries or veins, torsion of the spermatic cord, congestion or hyperdistention of the spermatic cord or seminal vesicles, orchitis, muscle strain due to running, jumping, horseback riding, straddling, and foot diseases may cause pain in either or both inguinal regions

Pain in the Hypogastric Region: It may be caused by disease of the bladder with urinary retention; diseases of the uterus or the prostate; by pelvic cellulitis; by periostitis or other disease of the pelvic bone; by enteroptosis; and may occur during labor and abortion; in chronic constipation; in tumors of the rectum; in transverse myelitis, and in inflammatory diseases of the lower spine

F. Pain in the Rectum

It may result from ischiorectal abscess, hemorrhoids, fissures, ulcerations, and stenosis of the rectum It may also occur as the result of carcinoma, polypi and other affections of the rectum as well as from foreign bodies and fecal impactions. The various types of diarrhea and other local irritation, such as may be caused by irritating foods, may cause pain and burning Local infections of the anus may cause severe pain and itching

G. Backache and Spinal Column Pain[1]

Pain along the spine or in any part of it is a common complaint and may be due to many causes, such as disease and deformity of the vertebrae, the articular surfaces, the ligaments or the spinal muscles. It may be due to muscle strains, skin sensitivity, jarring, sprains, faulty posture and flat feet. Backache may also occur as a reflex phenomenon resulting from disease of the thoracic and intraäbdominal viscera, and it often accompanies systemic disease, acute and chronic infections and functional or organic nervous diseases. The pain may be sharp, dull, aching or just a tired or draggy feeling; it may be constant or intermittent, and may become aggravated on motion or stop when at rest. The pain may affect the entire spinal column and radiate to other structures or it may affect any portion of the spine; it may be unilateral or bilateral, or it may be strictly circumscribed. In attempting to discover the cause of backache it is important to elicit a definite history as to the method of onset, location of the pain, points of tenderness and the influence of motion, and also information as to previous diseases and accompanying ailments.

Pain in the Cervical Region: This is characterized by stiffness of the neck, limitation of head movements from side to side or forwards or backwards. The pain may be referred over the occiput, to the clavicles or down one or both arms. Occasionally there may be difficulty in swallowing Among the causes of pain in this region are:

Diseases of the Bone: These include cervical spondylitis due to tuberculosis or osteomyelitis; arthritic changes particularly in the fifth, sixth and seventh cervical vertebrae; rheumatic disease; fractures; scoliosis of the cervical spine; Paget's disease; subluxations of the atlas or axis; congenital deformities, cervical rib, and carcinoma of the cervical vertebrae or of the occiput.

Sprains: These are due to violent trauma which may cause rupture of

[1] SEE ALSO· pp 861 and 958.

The history of pregnancy and the presence of a fluctuating mass in the *cul-de-sac* are confirmatory signs. The temperature may be subnormal.

Ovarian Cyst (twisted pedicle): Such a cyst will cause sudden severe pain in either inguinal fossa and in the lower abdomen. The presence of a tender mass is of diagnostic importance.

Inguinal Hernia, if incarcerated or strangulated, will cause acute severe pain, often followed by shock with signs of acute intestinal obstruction such as abdominal distention, constipation, and vomiting which at times is stercoraceous.

Cryptorchism: When the undescended testicle becomes inflamed while in the canal it will cause acute severe pain on the affected side in that region and may be referred to the lower abdomen. A similar pain may be caused by *inflammatory bubo* and by *acute epididymitis.*

Acute Diverticulitis: The pain comes on suddenly in the left iliac fossa and is similar to that of appendicitis found on the right side. Rectal examination will elicit tenderness in the left iliac fossa.

Acute Pyelitis: This may cause pain in either iliac fossa. The pain is acute and is accompanied by tenderness in the affected loin, by chills, fever, frequent and painful urination, pyuria and often bacilluria.

Suppurative Periostitis of the Ileum: The condition is characterized by severe aching pain at or near the site of infection, which is worse during the night. Palpation may reveal local swelling and tenderness.

Psoas Abscess: The pus burrowing beneath the psoas muscle causes distention and tension which produces a throbbing pain on the affected side, which is worse at night. There is also *pain on movement of the leg.* The physical examination will reveal tenderness, fullness and often fluctuation. There is flexion of the thigh with angular deformity of the hip.

Ulcerative colitis, tuberculosis of the Cecum, Carcinoma of the Colon and Fecal Impaction: These conditions may cause pain in either iliac fossa. X-ray studies will usually reveal the seat of pathology.

Regional Ileitis: The pain is colicky, it is felt around the umbilicus and in the right lower quadrant of the abdomen. There is associated abdominal distention and diarrhea with watery, occasionally blood stained, stool. This may alternate with constipation. A sausage-shaped mass may be palpable in the right iliac fossa.

Lobar Pneumonia: It may cause referred pain to the right iliac fossa and in children is often mistaken for acute appendicitis. In these cases there may be severe hyperesthesia of the skin, but deep pressure does not aggravate the pain and muscle rigidity may be absent. A thorough examination of the lungs should therefore be made in all cases of severe right-sided abdominal pain.

Typhoid Fever: Tenderness, gurgling, rigidity and, at times, abdominal pain are among the symptoms present sometime during the course of typhoid fever.

Perforation of an Ulcer: It will cause *acute pain* and will be followed by signs of peritonitis.

Iliocecal Tuberculosis: This may at times cause pain in the right iliac fossa; it is usually accompanied by diarrhea and other signs of tuberculosis.

Various Other Causes: Pain in the iliac fossae may also be referred from

proper shoes and unequal length of the lower extremities

Reflex causes for lower back pain are many Among the commoner causes are: Kidney affections such as nephritis, renal infarcts, pyelitis, pyelonephritis, perinephritic abscess, renal tumors and malignancies, hydronephrosis, torsion of a ureter, *renal tuberculosis and adrenal* tumors, gastrointestinal disease, such as gastric or duodenal ulcers, gastric carcinoma, carcinoma of the colon, spastic and ulcerative colitis, visceroptosis, Glenard's disease, chronic intestinal obstruction, fecal impaction, chronic appendicitis and mesenteric thrombosis; biliary tract disease such as cholecystitis and cholelithiasis; hepatomegaly; pancreatic disease; certain of the blood dyscrasias; splenomegaly, aneurysm of the abdominal aorta; disease of the ovaries and uterus, or disease of the prostate

Pain in the Sacroiliac and Coccygeal Regions: It may be caused by disease of these bones or their articulations; by tumors, fractures and various types of arthritis; and reflexly from disease of the pelvic organs, the bladder, the rectum, the prostate and the posterior urethra. It may also result from ischiorectal abscess, *infections, granulomata* affecting the peroneum, pilonidal cyst, peroneal fistulae and spina bifida.

Pain Anywhere Along the Spine or in the Back: It is often found in the various organic nervous diseases such as spinal meningitis, myelitis, poliomyelitis, multiple sclerosis, syringomyelia, tabes dorsalis, tumors of the spinal cord and vertebrae, and spinal cord hemorrhage Pain along the spine and in the back is a frequent complaint in neurasthenia, hysteria, traumatic neurosis (railway spine), flat feet and exhaustion.

VI. Pain in the Bones and Joints[1]

A. Pain in the Bones (Ostalgia)

Pain in the various bones of the body may be generalized or localized *Localized pain* may be due to conditions of the bone in which the periosteum or the endosteum or both are involved. These may result from periosteal lesions, traumatisms, neoplasms, cystic degenerations, inflammations and fractures

Generalized pain may be due to osteomalacia, new growth and systemic disease. The character of the pain may be sharp and of sudden onset as in toothache and osteomyelitis, or it may be dull and aching as in syphilitic lesions Nocturnal ostalgia occurs in syphilis, tuberculosis of the bones, confined subperiosteal pus, and often in typhoid fever.

[1] SEE ALSO p. 723.

Local Bone Pain

Periosteal Lesions: Periosteal lesions causing pain are usually associated with inflammation and may be due to traumatism such as a bruise or a partial bone fracture, or it may be caused by subperiosteal hemorrhage, inflammation, or infection. The pain is usually localized; the area affected is raised and, in addition to the sharp pain constantly present, there is exquisite tenderness on palpation or on pressure. There is also severe pain on motion. In acute inflammation there is usually local redness, heat and swelling In the presence of suppuration, a fluctuating area may be palpable. Subperiosteal hemorrhage may cause pain because of subperiosteal pressure.

strands of muscle or of ligaments, sudden twisting of the head, straining of the head or neck against resistance

Strains: These are caused by holding the head in one position over a long period of time, such as may be found among needle workers, typists, proofreaders, microscopists, swimmers, and others who have a tendency to tire or strain their cervical muscles

Reflex Causes: In this group may be included retropharyngeal abscess, Bezold's abscess (an abscess below and behind the mastoid), aneurysm of the circle of Willis, affection of the second and third molars, eyestrain, certain types of headache; and affections that cause nuchal rigidity such as meningitis, tetany, tetanus, dengue, influenza and exposure to "drafts and colds." Torticollis, certain neuroses, suppurative thyroiditis, adenitis, adenolipomatosis and other conditions that interfere with head posture and cause strain of the muscles of the neck or of its blood vessels may cause transient, intermittent or constant pain.

Pain in the Thoracic Spine: It may be associated with spinal rigidity and deformities. The pain may be referred to the arms, the chest or the abdomen If the spinal nerves are involved the radiation of the pain is along the distribution of the involved nerves. The more common causes for pain in the dorsal region are

Skeletal Changes: These are osteoarthritis, Pott's disease, spondylitis, Paget's disease, Kummell's disease, spinal fractures, spinal deformities, carcinoma or sarcoma of the spine or cord, dislocation of the spinal vertebrae, injury or inflammation of the nucleus pulposus

Muscular and Ligamentous Causes: These are strain due to faulty posture and hyperactivity of the arms, such as is found in weavers, cigar makers, pressers, writers, swimmers, etc.

Reflex Causes: These may be referred from the diaphragm, gallbladder, pancreatic disease, intestinal obstruction, gastric ulcer, or carcinoma, fractured rib, intercostal neuralgia, empyema, carcinoma of the lungs, pulmonary emphysema, asthma, mediastinal neoplasm, and thoracic aneurysm.

Pains in the Lumbosacral Region (lumbago): Pain in the lower back is much more common than pain in the upper spine chiefly because of the great mobility of the lumbar spine and the anatomic relationship between the fifth lumbar and the upper sacrum The pain may be severe or dull, and may cause rigidity of the spine with spasticity of the spinal muscles. The pain from the spine may be referred to the abdomen; along the entire spine and down the thighs and legs, or along the course of the affected spinal nerves. The spine as well as the body as a whole is held rigid, as motion, change of posture or attempts at walking may aggravate the pain

Pain in this region may be due to osteoarthritis, sacralization, spondylithiasis, prolapsed nucleus pulposus, infective arthritis, tuberculosis of the spine, hypertrophic and atrophic arthritis, neoplasms, and suppurations. It may be caused by sprain of the articular surfaces, the ligaments or by rupture of muscle fibers and ligaments which may be due to violence, sudden motion, lifting of heavy loads or other traumata. Pain in the lower back may also be due to strain caused by prolonged effort against resistance, such as carrying heavy burdens, by prolonged stooping, assuming unnatural or unaccustomed postures; and by flat feet, im-

B. Pain in the Joints

Joint pains may be divided into two classes: (*a*) Arthralgia or neuralgic pain, in which structural changes may or may not be present. (*b*) Arthritis or organic pain, in which there are structural changes in any of the tissues comprising the joint such as the bones, cartilage, synovial membrane, capsule, muscles, tendons and skin This may be acute or chronic.

Arthritis: The pain in arthritis is aggravated by motion, jolting, jarring and by pressure The affected joint is usually held at partial flexion which is the natural relaxed position during rest or deep sleep The pain is more severe in acute joint affections than in the chronic forms. Radiation of pain from the affected joint to distant parts is seen in but few instances, as in the following: Hip joint disease will cause referred pain to the knee and inner side of the leg. Shoulder joint disease may cause radiation of pain to the deltoid, trapezoid and the supraspinous fossa. Metatarsalgia or flat feet will radiate pain to the ankles and calf muscles When the nerves are impinged upon or are inflamed because of joint affections the pain may be referred to the final distribution of their sensory fibers. Pain and deformities of joints may also be secondary to nervous affections such as is seen in syringomyelia, amyotrophic orthopathies due to spinal lesions, and joint affections following neuritis.

Acute arthritis: This may be rheumatic, gonorrheal, septic, embolic, tuberculous, syphilitic, hemorrhagic, traumatic or gouty.

Chronic Arthritis: It may have an acute onset and eventually become chronic, or it may have an insidious onset and show evidence of chronicity from the start. Among these latter may be mentioned gonorrheal, tuberculous, syphilitic, traumatic and hemorrhagic arthritis; gout; osteoarthritis; rheumatoid arthritis; Heberden's nodes, hyperparathyroidism (osteitis fibrosa cystica); sarcoma and carcinoma of a joint, Charcot's joints; chronic atrophic arthritis; chronic hypertrophic arthritis; hydatid cysts; bursitis; calcific deposits in joint spaces; displacement of articular cartilages; hemophilic, scorbutic and rachitic joints; pulmonary osteoarthropathies, Paget's disease; atrophic muscle disease; peripheral neuritis, and many other *chronic affections.*

C. Pain in the Upper Extremities

Pain in the Shoulder and Arm: Pain in the shoulder may be unilateral or bilateral. The pain may be reflected downward in the arm to the region of the insertion of the biceps, or it may descend to the forearm and occasionally to the fingers Pain in the shoulder or arm or in both may be due to: (1) Local injury to the bone, the shoulder joint, or the muscles of the shoulder and arm; to the blood vessels supplying the shoulder and arm, and to injury to the nerve supply (2) Disease of the bones and joints such as arthritis of the shoulder joint or of the cervical spine, multiple myeloma, osteitis fibrosa cystica, fractures, Charcot's joint, sarcoma of the upper end of the humerus, dislocations, tuberculosis of the bony structures of the shoulder, synovitis, subacromial bursitis, and calcific deposits in and around the joint. (3) Infectious causes producing vascular disease, neuritis, *neuralgia, thrombosis or* embolism of the brachial or other arteries of this region. (4) Reflex causes such as angina pectoris, coronary thrombosis,

Fracture: A fracture of a bone will cause pain either during motion when the fragments are disturbed, or during excessive callous formation when sensory nerve filaments become entangled. Fracture of a bone may be caused by traumatism or may occur because of decalcification.

Neoplasms: Bone tumors, malignant or benign, will cause pain only when the periosteum or a nerve becomes involved. Neoplasms occurring in a bone that is in motion such as a rib, spine, arm or leg will cause additional pain because of interference with muscular movements or because of pressure against pain-sensitive tissue or a nerve.

Infections: Infection of a bone may be caused by extrinsic trauma or by intrinsic infection. Extrinsic infection will show signs of inflammation Intrinsic infection may be caused by tuberculosis, syphilis, streptococcus, staphylococcus, or other infectious microorganisms Intrinsic infection of the bones may occur with pneumonia, typhoid fever, malaria or other diseases

Osteomyelitis: Osteomyelitis is an inflammation of the cancellous tissue and bone marrow. It may be of bacterial origin or it may occur in leukemia, Hodgkin's disease, and occasionally no definite cause is discoverable The pain occurs suddenly and is most intense During the early stages there are no external manifestations of inflammation other than an intensification of pain on pressure and fever Later the inflammatory process affects the cortex of the bone, the periosteum and the surrounding soft tissue. Osteosarcoma, gumma, osteoperiostitis and tuberculosis will cause localized pain over the lesion, and often over the entire affected bone.

Generalized Bone Pain

Osteomalacia: This is a chronic softening of the bones. It occurs most frequently during pregnancy. The long bones, the ribs, the pelvis, or the spine may become affected. This may cause pain on walking, deep breathing, bending or squeezing the affected part.

Osteitis Fibrosa Cystica (*Hyperparathyroidism*): This condition may, during the early stages, cause generalized pains and may therefore be mistaken for rheumatism. Later, when bony cysts form and fractures occur, the pain may be localized over the affected parts

Myeloma, Chloroma, and Hand-Schuller-Christian's Disease: These cause decalcification of bone and are accompanied by pain in the affected areas.

Osteitis Deformans (*Paget's Disease*) This frequently causes pain in the extremities and in the back and is probably due to the abnormal angulations on the pressure-bearing parts of the abnormal bones

Scurvy: Among the early symptoms of scurvy are tender shins.

Hydatid Cysts of the Bone: They are usually accompanied by periosteal pain.

Periarteritis Nodosa: *This often* causes severe pain in the extremities or over the ribs.

Pain in the vertebrae may be caused by Pott's disease; erosion of vertebrae by carcinoma or aneurysm, by sacralization or by fractures; also by disease of the intervertebral discs; by prolapsed nucleus pulposus; by spondylitis, and by painful conditions of the muscles of the back.

Generalized aching pains in the bones are experienced in dengue fever, influenza, etc.

locations, flat feet, articular rheumatism, *osteoarthritis, diabetic gangrene or gangrene* from other causes, endarteritis obliterans, thromboangiitis obliterans, Raynaud's disease, erythromelalgia, *arteriosclerosis, achillodynia, the various* types of dactylitis, and hallux valgus, varus, equinus or rigidus

VII. Miscellaneous Causes of Pain

A. Nerve Pain and Tenderness[1]

The two classifications of pain along the nerve trunks or their terminal distribution are neuralgia and neuritis. Behan states that "the distinction between neuralgia and neuritis are quantitive rather than qualitative It is largely a matter of degree. A severe neuralgia may be termed a neuritis; a mild neuritis a neuralgia."

Neuralgia: This is defined as an affection of the sensory nerves characterized by intermittent severe lancinating or darting pain along the course of the nerve or its various distributions The overlying skin is sensitive and there are tender points corresponding to the locations where the cutaneous branches of the nerve are given off from the deeper structures Deep pressure is generally less painful than superficial palpation

Etiology: Neuralgia may arise from exposure to cold, infections, toxemias, trauma, pressure, vitamin deficiency diseases, diabetes mellitus, various poisons, rheumatic and gouty diathesis and from infectious diseases The commonest distributions of neuralgia are (*a*) trigeminal (tic douloureux) and other facial

popliteal branches; (*c*) intercostal, any of the intercostales may become affected; if the ganglion is involved herpes zoster may appear; (*d*) brachial, the pain may be along the courses of the brachial, subclavicular or cervical trunks and their distributions. Other distributions may be along the circumflex lumboabdominal, crural, visceral, cardiac, or any of the sensory nerve trunks and ganglia. The affection of the nerve may be accompanied by paresthesia, local anesthesia, sympathetic nerve features, muscular atrophy, spasms and vasomotor changes.

Neuritis: This may be defined as an inflammation of a nerve. It may affect a single nerve (local neuritis) or a number of nerves (multiple neuritis); and it may be acute or chronic. The inflammation may be interstitial or parenchymatous.

Etiology: Neuritis may result from traumata, exposure to cold, local and general infections, pressure, arteriosclerosis, toxins, metallic poisons such as lead, arsenic, bismuth, etc; and it may occur in diabetes mellitus, beriberi, deficiency diseases, alcoholism, rheumatism, tabes dorsalis and senility. The most outstanding symptom of neuritis is pain along the course of the nerve and its distribution. The pain is burning or boring in character. It is aggravated on movement of the affected part and during the night The nerve is extremely tender to pressure Other findings are anesthesia, paresthesia, wasting and often paralysis, and the *disappearance of the reflexes of the affected parts. The skin over the affected* part becomes atrophied and glossy and occasionally it may become thickened.

Sciatica: This is a term often applied to pain along the distribution of the sciatic nerve. Sciatica should be classified as primary and secondary.

Primary or True Sciatica: This is probably a neuralgia of the sciatic nerve caused by inflammation of the ganglia or of the perigangliar tissue. The exact cause is as yet unknown.

[1] See also p 855.

pericardial effusion, mediastinal tumor or aneurysm, diseases of the diaphragm, diseases of the gallbladder, cholelithiasis, cancer of the breast, pleurisy, pulmonary tuberculosis, calcified cervical glands and to tumor of the apex of the lung (sulcus tumor). (5) A variety of causes such as cervical rib, scalenus anticus syndrome and congenital deformities

Scalenus Anticus Syndrome and Cervical Rib: The symptoms of scalenus anticus syndrome and cervical rib are similar; both are due to neurocirculatory compression. An x-ray examination of the shoulder will reveal the presence of a cervical rib, while the diagnosis of the scalenus anticus syndrome is inferred from the clinical manifestations. There is pain in the shoulder and arm which is referred with varying intensity down the arm The pain is frequently associated with cramps, numbness and tingling in the hand or fingers. Often there is also coldness and apparent atrophy of the hand with areas of paresthesia. The pain is aggravated and the pulse becomes weaker by exercise, by adduction of the arm, by pressing forward of the shoulder, by pressing against the scalenus anticus muscle and when the chin is hyperextended and rotated towards the side opposite to the pain. This is due to the impingement of the subclavian artery and some of the cervical plexus nerves by the scalenus anticus near its insertion in the anterior third of the first rib.

Scaleniotomy near its insertion will relieve the symptoms and signs.

Pain in the Elbow: This may be caused by fractures, suppurations, trauma and other joint affections

Pain in the Wrist and Hand: This may be caused by fractures, sprains, occupational neurosis, gout, acroparesthesia, erythromelalgia, Raynaud's disease, thromboangiitis obliterans, various bone diseases, tuberculous dactylitis, rheumatic fever, various other types of arthritis, and also tumors such as sarcoma, chondroma, carcinoma, neurofibroma, and the various types of neuritis.

D. Pain in the Lower Extremities

Pain in the Hip Joint: This pain may be due to rheumatic fever, the various arthritides, traumata, dislocations, intracapsular fracture, or fractures of the structures entering into the formation of the hip joint, various bone diseases, osteitis fibrosa cystica, tumors, suppurations, tuberculous hip disease, iliopsoas bursitis, sarcoma, carcinoma, sciatica, disease of the lower spine, obdurator hernia, scurvy, appendicitis, and some of the neuroses

Pain in the Thigh: This may be caused by hip joint disease, sciatica, fractures, tumors, abscess of the thigh, thrombosis or embolism of the thigh vessels and of the iliacs, various bone diseases, traumata, disease of the lower spine, scurvy, psoas abscess, obdurator hernia, tumors of the spinal cord, anterior crural neuritis or neuralgia, fecal impaction, nephrolithiasis and trichiniasis.

Pain in the Knee: This may be caused by trauma, dislocations, fractures of the bones forming the knee joint, fracture or dislocation of the patella, the various arthritides (particularly rheumatic, tuberculous, gonorrheal, and osteoarthritic), dislocation of the semilunar cartilage, floating cartilage, prepatellar and interpatellar bursitis, suppurations, popliteal aneurysm, hip joint disease, fracture of the femur, disease of the feet (flat feet, corns, bunions, and metatarsalgia and improper shoes); also syphilitic arthritis, various bone diseases, intermittent hydrarthrosis (housemaid's knees), purpura hemorrhagica, hemophilia, osteitis fibrosa cystica and scurvy

Pain in the Feet and Toes: This may be caused by injury, frostbites, corns, calluses, bunions, fractures, dis-

insect bites; (*b*) *vesical,* due to acute cystitis, trigonitis, vesicle calculus, tuberculosis, cancer, ulcerations, foreign bodies, papilloma of the bladder and acute urinary retention; (*c*) *prostatic,* resulting from acute or chronic prostatitis, prostatic hypertrophy, carcinoma of the prostate, prostatic abscess *Referred pain* to the penis may result from nephrolithiasis, orchitis, sacroiliac disease, inflammation of the perineum, rectal carcinoma, hemorrhoids, rectal fissures and occasionally from acute appendicitis, particularly in retrocecal appendicitis.

Pain in the Testes: It may result from injury or disease. It is found in the various types of orchitis, epididymitis, *torsion of the cord, varicocele,* hydrocele, malignant tumors and tuberculosis; also in disease of the prostate, disease of the lower vertebrae, inguinal hernia, inflammation of the spermatic cord, nephrolithiasis, excessive venery, and mumps

Pain, Itching and Swelling of the Vulva: These symptoms may occur in local inflammations due to injury, infections, Bartholin's disease, carcinoma, tuberculosis, syphilis and granulomata, also in chancre, chancroid, lupus, condylomata and various skin affections. It may also occur in kraurosis vulvae, eczema, diabetes, herpes, during the menopause and in the various neuroses

D. Itching (Pruritus)

Itching is a peculiar sensation perceived by the skin and mucous membranes which is satisfied by scratching. It may be due to local irritation, systemic disease or allergic reaction.

Local Itching: Local itching may be caused by foreign bodies or other injuries, it is also found in hay fever, measles, nasal obstructions and eczema of the eyelids. Pruritus ani and vulvae may result from parasites, worms, local inflammatory conditions, dermatitis hives, irritating discharges, atrophic changes, toxic conditions such as diabetes, nephritis, cholemia, and during the menopause.

This may also be caused by pediculosis, scabies, dermatophytosis, various local skin diseases, frostbites, insect bites, local irritations due to sunburn, x-ray burn, scalding and other types of burns (during the healing stage) and by local interference with the circulation or innervation of a part.

General Itching: This is seen in most types of jaundice It is particularly prominent in pancreatic disease, gallbladder disease and in other types of obstructive jaundice. It is also found in diabetes mellitus, exophthalmic goiter, in various general skin diseases such as prurigo, lichen, eczema, seborrhea, mycosis, and in diseases in which there is sweating and desquamation; also in general urticaria, poison ivy and other irritations Various foods and drugs may cause itching of the skin though signs of urticaria be absent In morphinism it is a prominent sign.

Itching may also occur in the various neuroses It is at times present after a warm bath, after disrobing, particularly

bugs or other vermin. Occasionally the desire to scratch is brought on by seeing some other person scratching.

Itching, either local or general, is a common allergic manifestation. It is noted in the various types of urticaria (SEE: p. 927), in prurigo and in atriplicism.

Atriplicism is due to poisoning with atriplex littoralis. The young shoots of

is common among the poor of northern China who eat this plant because of food scarcity.

Symptoms: There is severe burning pain in the lumbosacral region, the hip joint and along the posterior aspect of the thigh, the calf muscles and at times in the outer surface of the foot. There is also tenderness along the sciatica nerve but seldom paresthesia, anesthesia, or muscle atrophy. Walking and extension of the leg are painful Flexion of the thigh without flexion of the leg is not possible. The tendo Achilles reflex is absent. Primary sciatica is not as common as secondary sciatica.

Secondary Sciatica: This may be a sciatic neuritis caused by disease of the spine, such as sacroiliac disease, spondylitis, tumor of the spinal vertebrae, fracture, prolapse or extrusion of the nucleus pulposus, spinal caries, etc. It may also be caused by tumors of the spinal cord and nerve roots, pelvic tumors, large prostate, and by inflammatory diseases of the hip, thigh and leg muscles, by flat feet, and by disease or deformities of the osseous structures of the spine, hip, thigh, leg or foot

B. Pain Due to Arterial Disease[1]

Diseases of the arteries such as arteritis, thrombosis, embolism and aneurysm usually cause pain in the parts of the body supplied by the affected arteries either because of interference with the circulation or because of injury to the tissue adjacent to them.

Arteritis: Painful conditions due to arteritis are intermittent claudication and other types of pain caused by endarteritis obliterans, thromboangiitis obliterans (Buerger's disease), diabetic gangrene, syphilitic endarteritis, aortitis, angiospasm, coronary disease, erythromelalgia, Raynaud's disease and periarteritis nodosa.

Thrombosis or Embolism: These conditions in any part of the body, except in the central vessels, cause severe pain. It is noted particularly in mesenteric thrombosis, splenic infarct, and coronary thrombosis, the pain being due to ischemia or anoxemia.

Aneurysm: This causes pain, first, by hyperdistention and injury to the arterial coat, and, second, by pressure against adjacent structures.

Pain Due to Disease of the Veins: This is noted in the various types of phlebitis and venous thrombosis The pain is usually felt at the location of the thrombus and along the course of the inflamed vein. There is also pain in the part supplied by the affected vein because of venous stasis and the resulting gangrene.

Pain Due to Interference with the Blood Supply of a Part: This may be caused by an overabundance of blood such as is seen in acute inflammations where the pain is sharp, acute, aching or throbbing; and in passive congestion where the pain is dull and sometimes aching due chiefly to hyperdistention, or it may be due to a diminished blood supply causing anoxemia. The pain in Raynaud's disease is felt in the hands or feet and is due to contractions of the arterioles, thus causing anemia In purpura hemorrhagica the pain is caused by obstruction in the arterioles. Pain in an extremity caused by the application of a tight tourniquet is due partly to venous congestion and partly to lack of arterial blood The pain in angina pectoris and coronary disease is probably due to ischemia of the heart muscle

C. Pain in the Genital Organs[1]

Pain in the Penis: It may occur during micturition, or it may be unrelated to urination. The commonest causes for such pain may be (*a*) *urethral*, caused by acute urethritis (gonorrheal or otherwise), urethral trauma, stricture, calculus, chancre, cellulitis, carcinoma, tuberculosis, cavernitis, and

[1] SEE: *Peripheral Vascular Disease*, p 535

[1] SEE: *Genital Diseases, Female*, p. 694, and *Male*, p. 707

energetic but consumes more food than is required for his or her maintenance. This type has frequent headaches, tires easily, may have attacks of syncope, has hypotension, but normal basal metabolism and is subject to diabetes mellitus

Endogenous obesity is attributable to diminished oxidation. While the individual may or may not take in more food than he can utilize, the abnormality lies in the lack of dissipation of energy rather than in the excessive consumption of food

Several distinct types of obesity are recognized

Pituitary Obesity: Hyperpituitarism, as seen in acromegaly, gigantism, basophilism and in the less pronounced forms of hyperpituitarism usually produces the tall, plethoric type of obesity. Hypopituitarism, as seen in Frohlich's syndrome, and in the adult types produces girdle obesity; the abdomen is fat and pendulant, the ankles and wrists are rather small, the skin is of fine texture and the hair distribution is heterosexual

Hypothyroid Obesity: There is uniform distribution of rather firm, nonyielding fat, with fat pads over the supraclavicular and supraspinous regions. The forearms and legs are large and fat; the skin is often of leathery texture

Hypogonad Obesity: There is a general distribution of fat with large fat pads over the trochanteric regions The genitalia are poorly developed and the sex functions are poor or nil

Adrenal Obesity: The fat distribution is over the upper part of the body; the lower part of the body is usually thin and there is accompanying virility, hypersexualism and hypertrichosis.

Pineal Obesity: This type of obesity may occur in young boys They develop prematurely; they are plethoric, have increased musculature, increased stature, up to a certain age; they are quite stout and have hypersexual development. The condition is known as macrogenitosomia precox.

Cerebral Obesity: General rapid increase in fat distribution may occur in some tumors of the brain, in certain of the brain diseases, as encephalitis lethargica and in other encephalopathies

Other Forms: Obesity also occurs in *lipodystrophy* and in *thymic disease.*

Loss of Weight (Emaciation)

Loss of weight may result from insufficient food intake, inability properly to utilize ingested food, rapid expulsion of food from the stomach by vomiting or diarrhea, and excessive expenditure of energy.

Rapid Emaciation: This occurs in all acute febrile diseases, in chronic infections, in carcinoma, tuberculosis, diarrhea, dysentery, progressive vomiting, the various digestive disorders, scurvy, pellagra, marasmus, exophthalmic goiter (in spite of voracious appetite), diabetes mellitus, parasitic infestations, pituitary cachexia, anorexia nervosa, Addison's disease, general loss of appetite or inability to eat, dehydration, starvation, overwork and insufficient sleep.

Changes in the Appetite[1]

The appetite may be variable. It may be excessive (bulimia; polyphagia), perverted or capricious (pica), unsatiated even after a full meal (acoria), or decreased (anorexia).

Excessive Appetite: It is characteristic of diabetes mellitus, hypopituitarism, and of certain nervous disorders

[1] See also: p 634

CHAPTER V

Miscellaneous Symptoms

Edema[1] (Oedema)

Edema consists of an abnormal local or general accumulation of interstitial fluid.

Edema of the lower eyelids may be caused by disease of the eyes and by acute coryza such as is seen in acute cold or in hay fever. It may be among the early symptoms of nephritis; in such cases the edema is worse on arising in the morning and may disappear as the day wears on. In severe cases of nephrosis or in tubular nephritis the edema may spread to the entire face and later to the body Edema of the eyes may also be due to local inflammation as orbital tumors, facial injury, skin diseases and erysipelas Edema of the face and neck may occur in mediastinal tumors. Edema of the feet or legs may be due to local injury, tight shoes or excessive tiredness and is an early sign in right-sided heart failure The edema is worse during the day and evening (if the patient is active) and disappears in the morning after a night's rest Edema is an indication of interference with the venous circulation of a part.

General Edema: This may occur in heart failure, glomerular nephritis, nephrosis, anemia, trichinoses, salt retention, starvation and inadequate intake of proteins. The edematous parts usually pit on pressure. *Lymphedema* is caused by decreased lymphatic drainage, and the edematous parts do not readily pit on pressure.

[1] SEE p. 150 and Index

Increase in Weight (Obesity)

Increase in weight, if not due to natural growth, may be caused by edema, accumulation of fluids in the serous sacs, by pregnancy, tumors, cysts, and by the rapid accumulation of fat as found in the various types of obesity.

An excessive amount of fat generally distributed through the body is due to a *disproportion* between the amount of food ingested and the amount of energy dissipated. Obesity is generally classified as: (1) Exogenous obesity due to (*a*) the consumption of large quantities of *food or drink*, (*b*) *to diminished activity*, and (*c*) to a combination of excessive food consumption and low expenditure of energy; (2) endogenous obesity due to some pathologic disturbance of the fat *metabolism center or to disturbance* of some of the endocrines (SEE: p. 772).

Exogenous Obesity: It occurs in otherwise normal persons. The individual, child or adult, has a voracious appetite and consumes large quantities of fat-producing food There is one type who is energetic, plethoric, physically strong and active and is in good health, except that he or she may have a tendency to dyspnea on moderate exertion The food intake is enormous and is in excess of the amount of energy expended. Another type is one who is lazy, listless, complaining, who eats moderately large quantities of food but dissipates little energy. This type of individual is usually anemic, may complain of headache, tiredness, indigestion, constipation, backache, dyspnea, and cardiac palpitation. A third type is moderately

energetic but consumes more food than is required for his or her maintenance. This type has frequent headaches, tires easily, may have attacks of syncope, has hypotension, but normal basal metabolism and is subject to diabetes mellitus.

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be, if alkaline, from the esophagus; and, if acid, from the stomach.

Regurgitation may be a symptom in esophagitis, stricture or obstruction of the esophagus, and esophageal diverticulum. It may also occur in gastric ulcer, in dilatation of the cardiac end of the stomach, in cardiospasm, and in various neuroses

Vomiting

Vomiting may be acute or chronic. The term acute here designates the sudden occurrence of vomiting without a previous history of recurrent attacks. Chronic vomiting is defined as recurrent attacks of vomiting over a long period of time (SEE. p 635)

Acute Vomiting: It occurs in seasickness, car sickness, etc.; following the taking of a general anesthetic, or of certain foods, and emetic drugs such as apomorphine, ipecac, copper sulfate, zinc sulfate, antimony and other drugs; in psychic shock, fright, undue excitement, anxiety or disgust, and after the smoking of the first cigar or pipe of tobacco. Acute vomiting may also occur in acute appendicitis, acute intestinal obstruction, incarcerated hernia, acute peritonitis, acute gastritis, acute gastroenteritis, migraine, cholecystitis, cholelithiasis, acute hemorrhagic pancreatitis, nephrolithiasis, acute Bright's disease, uremia, acute alcoholism, hyperdigitalization, and after the administration of morphine

Acute vomiting is an important symptom in fracture of the skull, cerebral concussion, cerebral embolism and sinus thrombosis. It also occurs in yellow fever, acute yellow atrophy of the liver and other types of acute hepatic degeneration.

Chronic Vomiting: This is associated with diseases of the digestive tract, the nervous system, the endocrine system and with intoxications and various reflexes.

Diseases of the Digestive Tract: *Stomach:* Carcinoma, ulcer, achylia gastrica, pyloric stenosis of infancy, gastrectasis, chronic gastritis, pylorospasm, ulcerations of the esophagus, chronic gastrorrhea, hour-glass contraction of the stomach, syphilis and tuberculosis of the stomach.

Intestines: Chronic intestinal obstruction, carcinoma of the colon or of the small intestines, dysentery, ulcerative colitis, ulceration of the intestine, paralytic ileus, diverticulitis, regional ileitis, intestinal worms, pancreatitis, pancreatic cyst, and adenoma of the islands of Langerhans.

Liver: Cirrhosis of the liver, amyloid liver, Banti's disease, carcinoma of the liver, the bile ducts or the gallbladder, abscess of the liver and passive congestion of the liver.

Diseases of the Nervous System: Cerebral tumor, cerebral abscess, hydrocephalus, cerebral hemorrhage, cerebral syphilis, locomotor ataxia, pachymeningitis, pituitary cachexia, etc.

The various neuroses, hysteria, psychasthenia, neurasthenia, psychic and emotional disturbances and in some of the insanities, and in Raynaud's disease.

Diseases of the Endocrine System: During a crisis in exophthalmic goiter, myxedema or Addison's disease

Diseases of the Cardiovascular System: Congestive heart failure chronic myocarditis, coronary thrombosis, aneurysm of the abdominal aorta, Stokes-Adams syndrome, and mitral stenosis.

Loss of Appetite: It occurs in various chronic gastrointestinal diseases, in fevers, and in most acute and chronic diseases. It also occurs in some of the neuroses, in anorexia nervosa, etc.

Aversion to Certain Types of Food: This is found when on a monotonous diet, in diseases of the gastric intestinal tract, in some of the neuroses and insanities, during early pregnancy and in other conditions where an achlorhydria exists. An aversion to meat is often an early sign in carcinoma of the stomach

Gastrointestinal Symptoms

Heartburn (Pyrosis)

Heartburn is a burning sensation felt in the epigastrium, precordium and deep in the throat. This is usually associated with hyperacidity Hyperacidity may be a symptom in acute and chronic gastritis, gastric ulcer, duodenal ulcer, gastrectasis, cholecystitis, and in spastic and ulcerative colitis. It may also occur in vagatonia, in highly neurotic individuals and during pregnancy. Occasionally heartburn may occur in achlorhydria

Time of Occurrence Heartburn occuring during feeding or soon thereafter, particularly when taking spicy foods or concentrated sweets, is a sign of inflammation of the esophagus and stomach. Heartburn two hours after meals that is relieved by taking food or soda is a symptom in duodenal ulcer. Heartburn occurring five or six hours after eating is often found in pyloric obstruction and in liver and gallbladder disease.

Nausea

Nausea is a peculiar sensation of impending vomiting felt at the infrasternal or suprasternal notch or in the throat and is often followed by vomiting. It may arise from various causes, such as psychic, reflex, nervous, gastrointestinal, toxic, etc.

Psychic Causes: Seeing revolting sights (operations, blood, vomiting); smelling nauseating odors; listening to gruesome, revolting or boring tales; and even the thought of certain unpleasant episodes

Reflex Causes: Irritation of the soft palate or retropharynx; eyestrain; diseases of the middle ear; Ménière's disease; *migraine; seasickness; car sickness*, pain; intestinal worms; ovarian disease, and pregnancy.

Nervous Causes: Hysteria; neurasthenia; psychasthenia; morning nausea in nervous and high strung children.

Gastrointestinal Causes: Cholecystitis; duodenitis; achlorhydria; chronic gastritis; acute gastritis; carcinoma of the stomach (an early symptom); pyloric obstruction; gastrectasis; cirrhosis of the liver; colitis; obstipation; toxic gastritis following an alcoholic debauch; or food poisoning.

Toxic Causes: Eating of fatty, greasy or spoiled food; overeating; uremia; pregnancy; hyperdigitalization; following the taking of drugs or poisons such as ipecac, opium, arsenic, mercury, phosphorus or lead; and allergic reactions

Various Diseases: Pellagra; diabetes mellitus during acidosis; acute pancreatitis; acute nephritis; pulmonary tuberculosis; exophthalmic goiter during crisis; Addison's disease; chronic myocarditis with passive congestion; mitral stenosis; and periarteritis nodosa.

Eructation (Regurgitation, Water-brash)

Regurgitation of small quantities of food without retching or vomiting may

air is most difficult. (*d*) Tumors, foreign bodies within the upper air passages, or stenosis of the bronchi from any cause may interfere with the entrance of air in the lungs or with its exit from the lungs. (*e*) Cardiac disease may cause an insufficient quantity of blood to be brought to the lungs for oxygenation as seen in acute or chronic myocarditis with cardiac decompensation. (*f*) Anemia or other blood dyscrasias may result in a scarcity of the oxygen-carrying corpuscles, hence a more rapid interchange between the alveolar air and the blood within the pulmonary circulation becomes necessary. (*g*) Fevers may require greater than normal amounts of air because of the increased metabolism. (*h*) Disease of the diaphragm, ribs and pleura may hinder proper lung expansion, thereby requiring more frequent lung action so as to bring the necessary amount of air in a given time. (*i*) Abdominal distention may crowd the diaphragm upwards and interfere with its motion, thereby hindering lung expansion. (*j*) Certain toxic states may cause anoxemia, to overcome it, respirations quicken (*k*) Disease of the nervous system or brain may interfere with respiratory centers (SEE: pp. 256, 466 and Index).

Hypopnea (*Slow Respiration, Oligopnea, or Bradypnea*)

Slow respiration is noted in intracranial pressure due to tumor, hemorrhage or cerebral concussion, and in basal meningitis. It is also found in diabetic coma, uremia, opium poisoning, chloroform narcosis and acute alcoholism. Large doses of chloral, aconite, antimony and the barbiturates may slow the respiratory rate sufficiently to cause cyanosis. Periods of hypopnea or apnea are seen in conditions that cause Cheyne-Stokes breathing, Biot's breathing, Stokes-Adams syndrome and occasionally, it occurs in those approaching death In hysteria and in certain convulsive states apnea may last for several minutes

Aphonia

Aphonia may be of four types: (1) Aphasia because of brain lesions; (2) disease of the vocal apparatus; (3) deaf-mutism, and (4) it may be a temporary condition due to neurosis.

(1) ***Aphasia Due to Brain Lesion:*** It may be caused by some organic focal cerebral lesion such as hemorrhage, thrombosis, embolism, tumor, abscess or gumma. The various types of aphasia depend upon the location of the lesion.

(*a*) When spoken words are not understood and cannot be repeated or written from dictation (cortical auditory aphasia) the lesion is to be found in the psychomotor center at the foot of the third temporal convolution (*b*) When the spoken words are not understood, cannot be repeated or written from dictation, but internal language (word thinking) reading (inaudible) and writing are not interfered with (subcortical auditory aphasia) the lesion is to be found in the first temporal convolution (*c*) When volitional speech is present, but reading or writing from dictation, or copy is impossible (cortical visual aphasia) the lesion is to be found in the gyrus angularis. (*d*) When language is understood, but the power of speech, and repeating of words are absent, and reading ability is lost (cortical motor aphasia) the lesion is to be found to extend from the temporal lobe to the cuneus (*e*) Sensory motor aphasia is a condition in which there

Diseases of the Hemopoietic System: Purpura, primary and severe secondary anemia, sickle cell anemia, and leukemia

Reflex Causes: Eyestrain, Ménière's disease, tuboövarian disease, pertussis, angioneurotic edema, allergic reactions, prostatitis, and cyclic vomiting in children.

Toxic Causes: Chronic glomerular nephritis, nephrosclerosis, chronic nephrosis, pregnancy, chronic alcoholism, and some of the vitamin deficiencies.

Diarrhea[1]

Diarrhea may be acute or chronic and the number of stools and their character vary according to etiology.

Acute Diarrhea: This may result from food and drug poisoning, from the use of various laxatives, and it may be brought on as an allergic phenomenon or by anxiety, nervousness and psychic disturbances. Acute diarrhea is found in enterocolitis, ileocolitis, ileitis, cholera morbus, Asiatic cholera, bacillary dysentery, acute amebic dysentery, sprue, pellagra, typhoid fever, influenza, mesenteric thrombosis and vitamin B and D deficiencies.

Acute infantile diarrhea occurs during the summer months and as the result of food deficiencies and indiscretions in diet; also as a result of various types of gastroenteritis.

Chronic Diarrhea: It occurs in chronic enterocolitis, ulcerative colitis, mucous colitis, tuberculous enteritis, sprue, celiac disease, chronic steatorrhea, carcinoma of the rectum, carcinoma of the pancreas, chronic amebic dysentery, nervous diarrhea, and in various chronic toxic conditions of the liver, the intestines and in parasitic infestations

[1] SEE ALSO: pp. 659 and 1031.

Constipation

Constipation may result from bad stool habits and from improper diet, insufficient liquids and sedentary habits. Constipation as a symptom in various diseases occurs in intestinal obstruction from any cause, strangulated hernia, neoplasms, strictures, mucous colitis, paralytic ileus, fecal impaction; also in lead poisoning, opium poisoning, visceroptosis, hemorrhoids, fissures and fistulae in the rectum and anus. It may occur constantly or intermittently in various chronic gastrointestinal diseases in gallbladder and liver diseases, in various nervous and mental diseases, in anemia and in various debilitated states

Respiratory Symptoms

Dyspnea and Orthopnea (Rapid, Difficult or Labored Breathing)

Dyspnea occurs because of insufficient oxygenation which the rapid respiratory rate attempts to supply. It may result from numerous conditions: (*a*) In health after exertion and in emotional states where an increased amount of blood is being used, more air is required and is thus being supplied; also in high altitudes where the air is rare, or in unventilated or stuffy places where the oxygen is insufficient, in order to supply greater quantities of air, a more rapid interchange between inspired and expired air takes place. (*b*) Pathologically dyspnea may be caused by diseases of the lungs which limit their air content such as consolidation of the lungs, lung tumors and suppurations, compression of the lungs by pleural effusions of air, serum or pus, or by mediastinal tumors or aneurysm. (*c*) Chronic emphysema and especially bronchial asthma may cause orthopnea because the exchange of

air is most difficult. (*d*) Tumors, foreign bodies within the upper air passages, or stenosis of the bronchi from any cause may interfere with the entrance of air in the lungs or with its exit from the lungs. (*e*) Cardiac disease may cause an insufficient quantity of blood to be brought to the lungs for oxygenation as seen in acute or chronic myocarditis with cardiac decompensation. (*f*) Anemia or other blood dyscrasias may result in a scarcity of the oxygen-carrying corpuscles, hence a more rapid interchange between the alveolar air and the blood within the pulmonary circulation becomes necessary. (*g*) Fevers may require greater than normal amounts of air because of the increased metabolism. (*h*) Disease of the diaphragm, ribs and pleura may hinder proper lung expansion, thereby requiring more frequent lung action so as to bring the necessary amount of air in a given time. (*i*) Abdominal distention may crowd the diaphragm upwards and interfere with its motion, thereby hindering lung expansion. (*j*) Certain toxic states may cause anoxemia; to overcome it, respirations quicken. (*k*) Disease of the nervous system or brain may interfere with respiratory centers (SEE: pp. 256, 466 and Index).

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Respiratory Symptoms

Dyspnea and Orthopnea (Rapid, Difficult or Labored Breathing)

Dyspnea occurs because of insufficient oxygenation which the rapid respiratory rate attempts to supply. It may result from numerous conditions: (*a*) In health *after exertion and in emotional states* where an increased amount of blood is being used, more air is required and is thus being supplied; also in high altitudes where the air is rare, or in unventilated or stuffy places where the oxygen is insufficient, in order to supply greater quantities of air, a more rapid interchange between inspired and expired air takes place. (*b*) Pathologically dyspnea may be caused by diseases of the lungs which limit their air content such as consolidation of the lungs, lung tumors and suppurations, compression of the lungs by *pleural effusions of air,* serum or pus, or by mediastinal tumors or aneurysm (*c*) Chronic emphysema and especially bronchial *asthma may* cause orthopnea because the exchange of

[1] See also pp 659 and 1031

at a bright light, particularly at the sun, and occasionally in some persons it occurs every morning after breakfast. There are also individuals who develop a paroxysm of sneezing after coitus and after a large meal, that is, when the stomach becomes overfilled with food or drink, or the colon is hyperdistended.

Cough *(See: p. 347)*

Cough is a sudden explosive expulsion of air from the lungs accompanied by a characteristic sound. It is a reflex response to some irritation in the retropharynx, larynx, the larger bronchi, lungs or pleura It may be caused by irritation, acute inflammation, passive congestion (as in heart disease) or by tracheobronchial-pulmonary obstruction Cough may also be due to nervousness and other extrapulmonary conditions.

The Character of the Cough: *Dry, Harassing, Nonproductive Cough:* These conditions are found in the early stages of bronchitis, the pneumonias, pulmonary infarcts, and in laryngitis, pharyngitis, tracheitis, elongated uvula, enlarged lingual tonsils, foreign body in the upper air passages, irritating dust or fumes, fractured rib, hilum tuberculosis, goiter, mediastinal tumor, aneurysm (brassy cough), Hodgkin's disease, pericardial effusion, neurosis, nasal polyps, pneumothorax, epiglottic ulcer, diaphragmatic paralysis, pharyngeal abscess, and esophageal diverticulum A slight, *dry, hacking cough occurring singly and* frequently repeated is often found in incipient pulmonary tuberculosis.

Moist, Productive Cough: It is found in the later stages of acute bronchopulmonary diseases, as in lobar and bronchopneumonia; in the later stages of acute bronchitis, and in subacute and chronic bronchopulmonary diseases, as in bronchiectasis, chronic bronchitis, whooping cough, foreign bodies in the lungs, lung abscess, gangrene of the lung, bronchogenic and pulmonary carcinoma, pulmonary tuberculosis, pulmonary actinomycosis, psittacosis, pneumonoconiosis, bronchial asthma, and the various suppurative diseases of the lungs and bronchi.

Paroxysmal Cough: It occurs in whooping cough Coughing spells at long intervals occur in bronchiectasis and in the presence of a pulmonary cavity resulting from gangrene, abscess, tuberculosis, or other causes. When the cavities fill with secretion or when there is change of posture a paroxysm of coughing is initiated. Cough occurring on exertion is found in chronic pulmonary fibrosis, tumor of the lungs, mediastinal tumors or aneurysm, pleural and pericardial effusions, pneumothorax and cardiac decompensation.

Short coughs occurring at frequent intervals and accompanied by watery, and often by serous frothy bloodstained expectoration, is a sign of pulmonary edema usually caused by acute heart failure or by acute pulmonary irritation.

Laryngeal Cough: This may assume various qualities such as croupy, hoarse, ringing, brassy or metallic; and is caused by direct or indirect laryngeal irritation. These types are found in laryngeal spasm caused by the inhalation of foreign bodies, i e, food, irritating gases, etc.; *in ulceration of the larynx or vocal* cords; in irritation of the recurrent laryngeal nerve as in aneurysm, intrathoracic goiter, abscess or tumor in the upper mediastinum, enlarged mediastinal glands, and esophageal malignancy.

Suppressed Cough: A voluntary attempt to suppress coughing is usually

is neither ability to recognize symbols or written words (visual aphasia), nor to speak or pronounce them (alexia, motor aphasia) (SEE: p. 842).

In right-handed persons the speech centers are in the left side of the brain, and in left-handed persons these centers are in the right side of the brain.

(2) **Aphonia Due to Disease of the Vocal Apparatus:** This is a condition in which there is an inability to speak aloud; the individual may be hoarse or may only be able to whisper.

This condition may be caused by the various types of laryngitis such as tuberculous, syphilitic, diphtheritic, suppurative or atrophic; and by acute and chronic catarrhal laryngitis caused by irritations, inhalation of irritating substances, straining or infections. It may also be due to disease, growths or dislocations of the vocal cords, edema of the glottis, foreign bodies in the larynx, benign or malignant tumors of the larynx, mediastinal tumors, thyroiditis, aneurysm of the arch of the aorta, chronic pharyngitis, apical tuberculosis and tumors of the apex of the lung, bulbar palsy, and many of the conditions that may cause irritation of the larynx or pressure upon the nerves controlling the larynx or the structures entering into the formation of sound.

(3) **Deaf-mutism:** This is congenital. Many of the deaf mutes may be taught how to speak or to utter sounds though their hearing ability remains nil.

(4) **Temporary Aphonia and Aphasia:** They may occur in the various neuroses, particularly in hysteria and occasionally in neurasthenia, psychasthenia and the various exhaustive diseases. They have also been noted following an epileptic seizure, during attacks of migraine and during sudden and severe fright.

Hiccough (*Singultus*)

Hiccough may be described as a peculiar high pitched grunting or clicking sound caused by the rushing of air through the glottis due to spasm of the diaphragm resulting from irritation of the phrenic nerve. Hiccoughing may continue for a brief period; it may be intermittent or it may continue for several days or weeks, both when awake or during sleep. It is usually accompanied by visible contractions of the epigastrium or upper abdomen.

Hiccoughs may be caused by overeating or imbibing too freely of alcoholic beverages, and by various diseases of the stomach, intestines, liver, gallbladder, pancreas and kidneys. When it occurs in uremia and peritonitis it is a grave symptom. Hiccoughs may also occur in disease of the meninges of the brain, and in hysteria, exhaustive diseases, diaphragmatic pleurisy, gangrene of the lungs, cardiac decompensation and in many toxic states. Occasionally hiccoughs may appear in epidemics either associated with symptoms of influenza or encephalitis, or in the absence of any symptoms and signs of disease. It may last from several minutes to several days.

Sneezing (*Sternutation*)

Sneezing usually results from irritation of the nasal mucosa by dust, gases or other substances, or by tickling. It is found in acute rhinitis, nasal polyps, acute coryza and hay fever; in the neuroses; as an allergic reaction; in deflected septum, and when foreign bodies are lodged in the external ear canal pressing against the tympanum. Reflex sneezing may occur when a person looks

therefore believes that he has not slept at all; and the other type in which the patient sleeps very little or not at all Often the patient may have difficulty in falling asleep, or he may sleep soundly the early part of the night and awaken during the early morning hours.

Insomnia may be caused by pain, frequent urination, diarrhea, impacted colon, cough, dyspnea, itching and other physical irritants. It occurs in various nervous states (the neuroses), also in overwork, brain fag, excitement, joy, grief, and other emotional states. Insomnia may also be caused by various drugs such as caffein, tea, coffee, strychnia, belladonna, benzedrine and other sympathomimetics. Sleeplessness is common in some of the acute febrile diseases, particularly in lobar pneumonia It is found in hyperthyroidism, arteriosclerosis, some of the severe anemias, cardiac decompensation, severe hypotension, cerebral syphilis, delirium tremens and other toxic states, in some of the psychoses and in the meningitides

Dreams and nightmares usually occur in neurasthenia, functional neurosis, prolonged worry and excitement, in cardiac disease, asthma, acute indigestion, constipation, partial wakefulness, and when assuming certain positions in bed Dreams may also be cultivated as a habit, and certain drugs may cause either pleasant dreams or nightmares

Vertigo (Dizziness, Giddiness)

(See: p. 850)

Vertigo may be functional or reflex and it may be organic. It is a subjective sensation of loss of equilibrium causing the patient a great deal of alarm. The sensation is known as objective vertigo when objects seem to be whirling or swimming around the patient; and as subjective vertigo when the patient feels as if he is whirling, sinking or rising while the surrounding objects are at rest.

Functional or Reflex Vertigo: This may be due to acute or chronic gastrointestinal disease, constipation, copious diarrhea, gallbladder disease, eyestrain, cerebral anemia, sudden release of cerebrospinal pressure after lumbar puncture, shock, severe hemorrhage, impacted cerumen in the auditory canal, arteriosclerosis, essential hypertension, extreme hypotension, seasickness, car sickness, swinging, aeroplane sickness, or it may result from *riding in any moving vehicle*, rapid turning of the body, looking down from great heights, hyperextension of the neck when looking upwards for an extended period of time, sudden change of posture, watching rapid movements of others, drug intoxication such as morphine and other opiates, quinine, salicylates, alcohol, tobacco (early users), belladonna, chronic interstitial nephritis; and it may occur in the neuroses such as hysteria, neurasthenia, psychasthenia, and neurocirculatory asthenia.

Organic Vertigo: This occurs as the result of definite lesions in the brain, the vestibular apparatus or the intracranial nerves Vertigo is a prominent symptom in cerebellar tumor where the vertigo is constant during walking, standing, sitting or lying down. In cerebral tumor the vertigo occurs in attacks and is accompanied by a feeling of uncertainty of equilibrium and confusion In cerebral syphilis the vertigo becomes manifested on effort; in general paresis vertigo is transient and may precede convulsions, hemiplegia or coma. In multiple sclerosis, vertigo may occur on arising, attempting to walk or on movement of the head. In oculomotor paraly-

due to pain as in pleurodynia, acute pleurisy, acute diaphragmatitis, broken ribs, intercostal neuralgia; during the early stages of acute bronchitis because of substernal soreness; and in peritonitis or other painful conditions of the chest, spine or abdomen; and also when the patient is too weak to cough.

Inability to Cough: In the presence of profuse pulmonary secretion inability to cough may be found in paralysis of the diaphragm, in bulbar palsy or other neurologic conditions, in extreme distention of the abdomen, and in extreme prostration.

To diagnose a disease merely by a cough is impossible. A thorough physical examination and other studies of the patient are necessary. It is also important to study the sputum grossly and microscopically (SEE p. 1033).

Weakness (Adynamia, Asthenia)

Weakness or loss of strength, also known as fatigue, lassitude, languor, exhaustion, tiredness, faintness, malaise, prostration, etc., is a prominent and often a distressing symptom in many conditions. It occurs temporarily after severe exertion or emotional strain, from insufficient food or drink, inadequate rest or sleep, exposures to excessive heat; during various fevers or other diseases; in diarrhea, vomiting or other gastrointestinal disturbances, during convalescence from acute diseases; and it may follow overindulgences in alcohol, tobacco, tea, coffee and venery.

Diseases in which marked weakness is a prominent symptom are: Addison's disease; hypoadrenia, myasthenia gravis; hypothyroidism; exophthalmic goiter; hypoglycemic states; diabetes mellitus; diabetes insipidus; pituitary cachexia; hypopituitarism; Cushing's syndrome, late stages of acromegaly; anorexia nervosa; malnutrition, vitamin deficiencies; gastrointestinal diseases such as ulcer, malignancy, colitis, the various diarrheas; the anemias and other blood dyscrasias; hemorrhage; chronic cardiac and pulmonary disease; nephritis; neurocirculatory asthenia; the various neuroses; hypotension, and various acute and chronic diseases.

Cardiac Palpitation[1]

Rapid heart action may be due to physiologic reasons, *e.g.*, running or other physical exertion; to psychic disturbances as anxiety, terror, fear, hilarity, neurosis, or other psychic and nervous disturbances; to fever (for each rise of 1° F. of fever there is an increase of ten heartbeats per minute); to certain types of shock, copious hemorrhage, exophthalmic goiter, neurocirculatory asthenia, cerebral concussion, heat exhaustion and conditions that will either paralyze the vagus or stimulate the sympathetics; to diseases of the heart, *e.g.*, paroxysmal tachycardia, auricular flutter, auricular fibrillation, acute myocarditis, pericardiac effusion, cardiac decompensation and other diseases of the cardiovascular system (SEE: p. 467); to drugs and poisons, *e.g.*, the various coal tar derivatives that cause myocardiac weakness such as acetanilid, phenacetin, amidopyrine; to other drugs as atropine, tobacco, caffeine, coffee, tea, strychnine, ammonia, alcohol; and to allergic reactions; overfeeding, and exhaustion.

Insomnia (Sleeplessness)

Insomnia may be of two types: One in which the patient awakens a number of times during sleep and is unaware of the periods in which he has slept, and

[1] SEE: Tachycardia, p. 510.

Tremors of the Eyelids, Etc.: Tremors are seen in hysteria and other neuroses when the *eyelids* are closed. *Tremor of the protruded tongue* is often found in typhoid fever. In the neuroses *coarse tremors of the hands,* feet or body are brought out voluntarily and during excitement; they disappear during rest or sleep.

Occupational Tremors: They may develop in any group of muscles that are subjected to chronic strain or constant use

Hereditary Tremors: These usually affect the head or arms; the tremors are fine, regular and rapid They become more pronounced during voluntary motion and are slight during rest

Chronic Arthritis and Chronic Muscle Wasting: They may cause intention tremors which cease when at rest

War Psychosis (shell shock) and Neurocirculatory Asthenia: They may cause general or local tremor during excitement or physical effort; this ceases during sleep

Muscle Cramps (Muscle Spasm)

Sudden severe tonic or clonic contractions of groups of muscles associated with severe pain and accompanied by temporary partial or complete paralysis may occur from overexertion of a particular group of muscles, interference with their blood supply or irritation of their innervation They may be toxic phenomena, or they may result from certain nervous diseases. Thus muscle cramps occur in swimmers, divers (caisson disease or bends), in occupational neuroses as in telegraphers, violinists, typists, etc., in thromboangiitis obliterans(Buerger's disease causing intermittent claudication), in tetany, spastic paraplegia, strychnia poisoning, heat exhaustion, alcoholic neuritis, hysteria, Asiatic cholera, and some of the diseases characterized by convulsive states. Myotonia (Thomsen's disease) is characterized by tonic spasms of the muscles when movement is attempted, it does not cause pain and is usually a hereditary disease. *Tonic preservation* or *tonic innervation* is a condition in which there is an inability to relax a group of muscles once they become contracted, as when an object is grasped and there is an inability to let go of it. This condition is due to a central lesion probably in the mid-frontal region (Mills).

Convulsions

Convulsions may be defined as paroxysms of involuntary and purposeless muscular contractions that may be limited to one or several groups of muscles or to the entire body They may be of variable duration and intensity. They may be tonic (slow and continuous) or clonic (rapidly alternating between contraction and relaxation); and there may be consciousness or unconsciousness Convulsions occur in the following conditions:

Epilepsy: In grand mal the convulsions are tonic and clonic and are preceded by a cry. The patient, when not in bed, falls to the ground. He is unconscious, may bite his tongue, froth at the mouth and lose sphincteric control. When the convulsion is over the patient falls into a deep sleep. In petit mal or jacksonian epilepsy a single group of muscles or one extremity may develop convulsions and there may be momentary unconsciousness. Epilepsy may be idiopathic, or may be caused by brain tumor or syphilis.

sis the vertigo appears when the gaze is turned towards the paralyzed muscle and it disappears when the paralyzed eye is covered or when the head is tilted so that the unaffected eye alone is in use. In labyrinthitis the vertigo is constant when standing, reclining or when the eyes are shut; it is accompanied by nystagmus, disturbance of equilibrium, nausea and vomiting In Ménière's disease the vertigo comes on in paroxysms; the patient often falls to the ground because it is almost impossible to maintain the erect posture; the vertigo continues in the recumbent position and the seizure terminates with nausea and vomiting.

It is often difficult to differentiate between reflex vertigo and the organic form. It is therefore important to evaluate the history and all the symptoms and signs associated with the attacks and those occurring between the attacks. Nearly all cases of vertigo are accompanied by a sense of panic, many have nausea and some have vomiting

Tremors *(See: p. 846)*

Tremors may be transitory or constant.

Transitory Tremors: They may occur because of excitement, fear or other *emotional stress; chills preceding fever;* exposure to cold; asthenia; excessive use of tea, coffee, tobacco, alcohol; and poisoning by mercury, lead, chloral, cocaine, morphine and other opiates and absinth

Constant Tremors: They may affect the hands, feet or the entire body and are characteristic of:

Paralysis Agitans *(Parkinson's disease)*: The tremor is constant and while the patient is at rest, it affects chiefly the upper extremities. The face is expressionless though the eyes are brilliant; the body is "set" and there is slowness in starting locomotion.

Senility: The tremor is first limited to the head and may later involve the whole body. The tremors are fine and are aggravated by voluntary motion and by excitement.

Encephalitis Lethargica *(Parkinson's type)*: The tremor is in the arms and legs; it is rather coarse and is continuous during rest.

Multiple Sclerosis: The tremor may affect the entire body and is brought out by attempted action (intention tremor); the tremor stops when at rest.

Progressive Lenticular Degeneration *(Wilson's disease)*: During the early stages the tremor is fine; it becomes more pronounced on physical or mental effort and may be voluntarily stopped for short periods. There may be progressive interference with swallowing and with speech, the consonants are slurred and the last syllables are dropped

General Paresis: The tremors are first noted about the face, lips and tongue. They occur at rest but are aggravated by motion such as attempted protrusion of the tongue or by attempting to speak.

Hemiplegia: The affected and weakened limb may have a Parkinson-like tremor which is aggravated by motion or excitement.

Intracranial Tumors: Those affecting the pons, crus, optic chiasm, the frontal lobes or the cerebellum, and other brain diseases may cause intention tremors.

Exophthalmic Goiter: This is characterized by fine tremors of the outstretched hands; occasionally it is accompanied by coarser tremors over the body

electric current. During the convulsions there is total loss of consciousness with severe tonic and clonic spasms of the muscles of the face, upper and lower extremities and of the trunk.

Fainting Attacks (Syncope)

In most instances syncope is a vasomotor phenomenon and may range in severity from drowsiness to periods of unconsciousness which may be momentary or may last for several minutes. Often the patient may be in a state of complete relaxation where volition is suspended though he may be conscious of his surroundings. It differs from coma which is brought about by definite pathologic conditions and causes complete unconsciousness Fainting spells are common among certain types of nervous individuals and are brought on by fright, excitement, grief, hilarity and other emotional states. Some individuals will faint at the sight of blood or at the sight of a surgical operation Occasionally it may come on after suddenly arising from sleep, particularly when there is an urge for a copious bowel movement or when there is a hyperdistended bladder with an urge to micturate Fainting is due to anemia of the brain in those who have neurovascular instability It is of little importance in young people, lowering the head below the level of the body will quickly restore the circulation providing the syncope is not caused by sudden severe hemorrhage Syncope is characterized by pallor of the face and lips, cold clammy skin, weak pulse and inactive pupils In old people syncope may be due to organic causes and is therefore serious Syncope may occur during the course of various diseases such as arteriosclerosis, chronic myocarditis, coronary thrombosis. Stokes-Adams syndrome, aortic stenosis, severe anemias, Addison's disease, Raynaud's disease, and it may also occur in heat exhaustion, hypoglycemia, after an injury and during hemorrhage.

Coma (Unconsciousness)

Coma is a state of unconsciousness from which the patient cannot be aroused until the cause of the coma is partially or entirely removed. During coma there is loss of consciousness, sensibility and motility. The reflexes are absent, and the swallowing of liquids when forced into the mouth is not possible. Coma occurs as a terminal phenomenon in many diseases, and also in many conditions that are not necessarily terminal It is therefore important to diagnose the etiologic factors responsible for coma

In examining a patient in coma the following routine should be followed: A brief history should be obtained from attendants when possible; the head of the patient should be carefully examined for signs of injury and for bleeding from the nose, mouth or ears; the odor on the breath should be noted; the state of the pupils should be observed; the reflexes, superficial and deep, should be elicited; the existence of paralysis, spasms or of flaccidity should be noted; the general appearance of the patient, the color, temperature and moisture of the skin, the type of breathing, and the condition of the pulse should be observed; and a urinalysis and blood chemistry should be done as soon as possible.

The commonest causes for coma are: (*a*) Cerebral hemorrhage, and other intracranial accidents; (*b*) uremia; (*c*) diabetes; (*d*) hypoglycemia; (*e*) drug poisoning; (*f*) severe alcoholism; (*g*)

Eclampsia: This occurs during pregnancy, or during or after labor. The convulsions come on suddenly and are most often clonic in character; occasionally they may be tonic The eyes roll upwards, the pupils dilate and there is twitching and distortion of the facial muscles. The convulsions spread rapidly to the extremities and to the body, which becomes rigid. The breathing is stertorous; there is frothing at the mouth and the face becomes congested. There may be several paroxysms separated by periods of coma

Uremia: The convulsions are epileptiform and recur rapidly. They may be jacksonian or general; the initial cry is absent The convulsive seizures may be preceded by headache, apathy, drowsiness and other cerebral symptoms. Following the convulsive seizures there may develop temporary blindness or deafness. The clinical features and laboratory examination of the urine and of the blood show characteristic findings.

Hypoglycemia: The convulsions are epileptiform and are usually localized to one side of the body. The patient is bathed in perspiration and the skin is clammy; the pulse is rapid and the breathing is rapid and shallow.

Tetanus: The convulsions are tonic and first affect the muscles of mastication (trismus) and then spread to the muscles of the back, causing opisthotonos; the body and extremities may become rigid (orthotonos), or the body may bend to either side (pleurothotonos) or forwards (emprosthotonos). The eyebrows may be raised and the angles of the mouth drawn out, producing the so-called *risus sardonicus.* The slightest irritation may bring on convulsive seizures. There is no loss of consciousness during the convulsive seizure; therefore they are attended by severe pain.

Hydrophobia (rabies): The spasms are usually limited to the muscles of deglutition and the larynx; swallowing causes painful spasms in the neck muscles. During the spasm the patient is hyperexcited and may become maniacal.

Tetany: The convulsive seizures are paroxysmal and may last from a few minutes to several hours. The spasms may affect the flexor muscles of the upper extremities alone, or the lower extremities alone, or the entire body may become affected. There is carpopedal spasm causing the obstetric hand or the claw hand. The toes may be hyperflexed and the feet are held in the talipes equinovarus position The thigh muscles are seldom affected. The head may be turned to one side and laryngismus stridulus may be present. The patient is conscious and the convulsions are painful

Infantile Convulsions: They may result from gastrointestinal disease; or they may occur at the onset of any acute infectious disease, teething, intestinal worms, thymus disease, rickets and spasmophilia. There is complete unconsciousness, rolling of the eyeballs, working of the jaws, and orthotonos.

Hysterical Convulsions: There is no complete loss of consciousness. The patient assumes various poses; there is fine blinking of the eyelashes; attempts to open the eyes are resisted; the sphincters are not relaxed. The convulsive seizures usually follow some emotional upset or when sympathy is demanded.

Other Causes: Convulsions are artificially produced in the treatment of various mental diseases by the intravenous injections of insulin or metrazol or induced by a properly controlled

Cerebellar Hemorrhage: If the fourth ventricle is involved this may cause coma with difficulty of respiration and swallowing.

Subarachnoid Hemorrhage: This seldom causes deep coma; there is nearly always nuchal rigidity and positive Kernig's sign; the deep reflexes are absent

Uremia: The coma is often preceded by headache, muscular twitching and occasionally by convulsions or by stupor. "Uremic frost" appears on various parts of the skin. There is Cheyne-Stokes breathing and a foul or uremic odor on the breath. Paralysis may or may not be present. The eye grounds may show retinal hemorrhages. The urine, if present, may show albumin and casts and the blood will show a high nitrogenous waste product content; the blood pressure is high

Diabetic Coma: It comes on slowly, it may be preceded by headache, apathy and drowsiness; the breathing is deep and sighing, the Kussmaul's air hunger type of breathing; the eyeballs are soft, and there is a fruity odor on the breath; the cheeks are flushed and the lips are cherry red There is marked dehydration and a rise in temperature. The urine contains sugar and acetone; and the blood may show a high glucose content while the CO_2 content of the alveolar air is low. The pulse is rapid and the blood pressure may be low (SEE: p. 799).

Insulin Shock: The onset of coma is sudden. The skin is cold and clammy and there is profuse perspiration; breathing is rapid and shallow. Plantar reflexes are elicitable Hypoglycemia may be as low as 50 mg or even lower.

Drug Poisoning: *In opium poisoning* the patient can usually be aroused; respirations are slow, 10 to 12 per minute; the pulse is slow and feeble; the skin is cold and clammy, and the temperature is low. The eyes will show pinpoint pupils both equally contracted There is an absence of localized paralyses.

In barbiturate poisoning the patient may be aroused for short periods during which he will mumble unintelligibly The pupils are usually dilated and there may be nystagmus. Abdominal and tendon reflexes are absent.

Alcoholism: The coma is not complete. The patient may be aroused. during which time he may mumble incoherently. The face is flushed or cyanotic; the pupils are equal and may be dilated. Respirations are of normal frequency though deep and noisy. The breath is alcoholic and is peculiarly sour or mawkish The odor on the breath should not be entirely relied upon for a diagnosis of alcoholic coma, since one who has been drinking alcohol may also develop a cerebral hemorrhage, or alcohol may have been forced on the patient in an attempt at resuscitation

Epilepsy: The coma usually follows epileptic convulsions and is of short duration There may be a bitten tongue and foam on the lips; the face is congested, the breathing stertorous, and the limbs relaxed

Sunstroke: The patient is wholly unconscious; the skin is hot and dry; the rectal temperature may exceed 109° F. The pulse is full and bounding; and the respirations are rapid, labored, deep and often stertorous. There may be convulsions.

Gas Asphyxia: The coma from gas asphyxia is associated with general cyanosis. The skin may be pale or have a cherry red color, or there may be cherry red blotches on an otherwise

epilepsy; (*h*) *sunstroke*; (*i*) gas asphyxia; (*j*) meningitis; (*k*) cerebral tumor or abscess; (*l*) freezing; (*m*) asphyxia; (*n*) Stokes-Adams syndrome; (*o*) hysteria, and (*p*) various endocrine and other disturbances.

Cerebral Hemorrhage: There is a sudden loss of consciousness with complete relaxation. The face may be pale or flushed; respirations are stertorous; the cheeks are inflated and the lips splutter during expiration. The pupils are either dilated or are unequal and inactive, except in pontine and ventricular hemorrhages when they are contracted. Hemiplegia is at first flaccid, later it becomes spastic; the Babinski sign is present on the affected side, at times on both sides. Hypertension may be present during a hemorrhage but falls when bleeding has stopped. The temperature may be normal or somewhat elevated. Hemorrhage into the ventricle when severe causes death within a few hours after the onset of coma; during coma the pupils are contracted or there may be conjugate deviation; the pulse is slow and respirations are labored.

Hemorrhage into the pons causes a rapid onset of coma; the pupils are contracted; respirations are slow; the temperature rises rapidly and may reach 103° to 104° F. or higher. There may be spastic movements of the limbs during the state of unconsciousness. Small hemorrhages into the pons may cause stupor in which the facial and ocular muscles as well as those of articulation and swallowing are involved. There may be unilateral paralysis to motion and sensation; at times there is crossed paralysis. During the early stages there is conjugate deviation away from the paralyzed side.

Cerebral Embolism: It may cause sudden loss of consciousness usually in a young adult; the pulse is rapid and the blood pressure is not changed; the temperature is normal. When consciousness is regained the existing paralysis may gradually disappear. There may be conjugate deviation.

Cerebral Thrombosis: If coma develops, it is of slow onset, usually occurs during the night in persons past middle life or in syphilitics. The temperature is normal; the pulse is rapid and weak, and there may be conjugate deviation.

Spasm of the Cerebral Arteries: There may be loss of consciousness. It occurs in the aged; the pulse may be slow; complete recovery may occur in from 12 to 48 hours.

Ingravescent Apoplexia: This is due to rupture of one of the branches of the external lenticular artery. The hemorrhage is at first in the external capsule. It subsequently breaks through the white matter into the lateral ventricle. The symptoms begin with headache, vertigo, vomiting, followed by hemiplegia, hemianesthesia, coma and death in a few days.

Fracture of the Skull: Coma may come on soon after or within 24 hours after the injury. There may be external evidence of trauma; the blood pressure is high and the pulse is slow. There may be edema of the retina and the escape of cerebrospinal fluid from the nose or the ears; nausea and vomiting may precede the coma Concussion of the brain may cause coma which, in the absence of hemorrhage, may last from a few minutes to several hours

Cerebral Tumor: Coma is of gradual onset preceded by headache. The presence of choked discs and other focal signs may help in the diagnosis.

pale skin. The respirations may be rapid and shallow or may be intermittent and gasping. The pulse is weak and rapid The odor of some of the gases may *cling to the patient.* Among the lethal gases are illuminating gas, automobile exhausts, coal gas, water gas, hydrogen sulfide (sewer gas), phosgene, mustard, etc.

Meningitides, Meningoencephalitis and Encephalitis Lethargica: These may cause coma. The etiology is determined by the history, febrile course, neurologic signs, cerebrospinal fluid examination and blood cultures

Brain Pathology: *Brain* abscess, tumor, multiple sclerosis, paresis, arterial spasm and acute *softening of the* brain may cause coma The diagnosis is based upon the history, local signs, cerebrospinal fluid findings and various neurologic findings

Freezing: This may cause total unconsciousness or coma. The history of the circumstances under which the patient was found may be sufficiently diagnostic, particularly if *there* are no external signs of injury or hemorrhage The *pulse and respirations* are *slow and* the general appearance of the patient is *that of tranquility* or as if in a *faint* The exposed portions of the body are *cold, stiff* and pale.

Asphyxia: When due to foreign bodies in the air passages, drowning, strangulation, suffocation, anterior poliomyelitis and pulmonary thrombosis asphyxia may cause coma which is diagnosed by the history, general lividity, distention of the veins in the neck, weak pulse, loss of phincteric control, and hemorrhage from the rectum, nose or other mucous surfaces.

Stokes-Adams *Syndrome:* *The* coma may be profound. The pulse is *extremely slow (ventricular)*; the auricular rate as noted in the vessels in the neck may be rapid; the breathing is stertorous, and there is general cyanosis. Epileptiform convulsions may occur during the state of unconsciousness.

Hysterical Coma: This is characterized *by the general appearance* of the patient; the assumed theatrical attitudes, *the flushed face; the normally* responding pupils; the resistance of the eyelids to attempted opening, and the upturned eyeballs. The pulse may be normal or somewhat rapid; respirations may be slow, normal or rapid, but are not stertorous. Coma or trance always occurs *before an audience and the patient* always chooses the spot upon which to *fall. Organic symptoms are absent.* The patient may be aroused when made to inhale irritating vapors such as ammonia or glacial acetic acid, or when pressure is made upon the supraorbital nerve or other sensitive spot.

Endocrine and Other Disturbances: *Coma may occur in tumor* of the islands of Langerhans *(hypoglycemia), in hemorrhage and tumor of* the adrenals, and in the pituitary *tumors; it may also occur in Addison's* disease, myxedema, exophthalmic goiter, tetany, hydrocephalus, and other grave toxic states (SEE: Chap XXVI, p. 755, and Index)

Special Symptoms of Mental Disease (SEE: p 892).

CHAPTER VI

Methods of Physical Examination

Physical examination may be defined as the act of ascertaining the condition of the patient's body by the aid of the special senses, supplemented by the use of such instruments as enhance the acuteness of these senses, *i e.*, the stethoscope, thermometer, sphygmomanometer, etc.

A *physical or objective sign* is one that can be seen, heard or felt by the examiner. These signs are sought for by five methods.

Inspection: Inspection is the act of examining a patient by the sense of sight, comparing the part under examination with one's mental picture of a similar healthy part, and one side of the body with the corresponding part of the opposite side. It is quite natural that inspection should be the first method of procedure in a physical examination, because the eye will recognize outward conditions long before the other senses can be brought into activity. Certain impressions are created by observing apparent trifles, which may prove valuable on further examination. Expert clinicians at times are able to make a diagnosis by apparent intuition, because they see and observe more closely than do others It is, therefore, of great importance to practice inspection thoroughly and systematically.

Palpation: Palpation is the act of examining an underlying organ by feeling with any part of the hand the overlying surface; and is usually the second step in a physical examination. Unfortunately, because of their eagerness to auscultate, many examiners too frequently neglect palpation and, as a result, their tactile sense is not as acute as it might be made if they practiced palpation at least as frequently as they do auscultation and percussion. As one grows older the sight may become dim, the hearing loses a great deal of its acuteness, but the tactile sense usually remains unaltered, and in many cases, it becomes even more precise. In order to be of value in a physical examination, palpation must be conducted systematically and with a definite object in mind In other words, one must know how to palpate and have a definite reason for so doing.

Percussion: Percussion is the act of striking or tapping the surface of the body in order to elicit such sounds as are produced by setting the underlying viscera into vibration. By percussion are elicited various sounds and degrees of resistance depending upon the nature of the tissue struck, *i e*, a solid substance when struck produces a dull or muffled sound, while an air-containing one gives rise to a clear or resonant sound. The proportion of air and solids in a substance determines its degree of clearness or dullness. The sound elicited by percussion enables one to distinguish the healthy from the diseased parts of the body.

Auscultation: Auscultation literally means the act of listening for sounds If a sound is produced outside of the body by striking upon the surface, directly or indirectly, the procedure is termed percussion. However, when lis-

TABLE III

Weight-Height-Age Table for Girls from Birth to School Age

Height (Inches)	1 mo	3 mos	6 mos	9 mos	12 mos	18 mos	24 mos	30 mos	36 mos	48 mos	60 mos	72 mos
20	8											
21	9	10										
22	10	11										
23	11	12	13									
24	12	13	14	14								
25	13	14	15	15								
26		15	16	17	17							
27		16	17	18	18							
28			19	19	19	19						
29			19	20	20	20						
30			21	21	21	21	21					
31				22	22	23	23	23				
32					23	24	24	24	25			
33						25	25	25	26			
34						26	26	26	27			
35						29	29	29	29	29		
36							30	30	30	30	31	
37							31	31	31	31	32	
38								33	33	33	33	
39								34	34	34	34	34
40									35	36	36	36
41										37	37	37
42										39	39	39
43										40	41	41
44											42	42
45												45
46												47
47												50
48												52

Weight-Height-Age Table for Boys from Birth to School Age

Height (Inches)	1 mo	3 mos	6 mos	9 mos	12 mos	18 mos	24 mos	30 mos	36 mos	48 mos	60 mos	72 mos
20	8											
21	9	10										
22	10	11										
23	11	12	13									
24	12	13	14									
25	13	14	15	16								
26		15	17	17	18							
27		16	18	18	19							
28			19	19	20	20						
29			20	21	21	21						
30			22	22	22	22	22					
31				23	23	23	23	24				
32				24	24	24	25	25				
33					26	26	26	26	26			
34						27	27	27	27			
35						29	29	29	29	29		
36							30	31	31	31		
37							32	32	32	32	32	
38								33	33	33	34	
39								35	35	35	35	
40									36	36	36	36
41										38	38	38
42										39	39	39
43										41	41	41
44											43	43
45											45	45
46												48
47												50
48												52
49												55

(By courtesy of The Children's Bureau, U. S Department of Labor)

TABLE I

AVERAGE WEIGHTS AND HEIGHTS AT VARIOUS AGES

MALE			FEMALE		
Age, Years	Average Height in Inches	Average Weight in Lbs	Age, Years	Average Height in Inches	Average Weight in Lbs.
6	46	48	6	45	45
7	48	53	7	47	50
8	50	58	8	49.5	57
9	52	64	9	52	64
10	54	71	10	54	72
11	56	78	11	56	80
12	58	88	12	58	90
13	60	98	13	60	102
14	63	113	14	62	114

TABLE II

SHOWING INCREASES IN WEIGHT AT VARIOUS AGES

MALE

Age Years	Year 52 Weeks		20 Weeks		Quarter 13 Weeks		Week	
	Lbs	Oz	Lbs	Oz.	Lbs	Oz	Lbs	Oz.
6	4.0	64	1.5	25	1.0	16	.077	1.23
7	5.0	80	1.9	31	1.3	20	.096	1.54
8	5.0	80	1.9	31	1.3	20	.096	1.54
9	6.0	96	2.3	37	1.5	24	.115	1.85
10	7.0	112	2.7	43	1.8	28	.135	2.15
11	7.0	112	2.7	43	1.8	28	.135	2.15
12	10.0	160	3.8	62	2.5	40	.192	3.08
13	10.0	160	3.8	62	2.5	40	.192	3.08
14	15.0	240	8	92	3.8	60	.288	4.61

FEMALE

Age, Years	Year 52 Weeks		20 Weeks		Quarter 13 Weeks		Week	
	Lbs	Oz	Lbs	Oz.	Lbs	Oz	Lbs	Oz
6	4.0	64	1.5	25	1.0	16	.077	1.23
7	5.0	80	1.9	31	1.3	20	.096	1.54
8	7.0	112	2.7	43	1.8	28	.135	2.15
9	7.0	112	2.7	43	1.8	28	.135	2.15
10	8.0	128	3.1	49	2.0	32	.154	2.46
11	8.0	128	3.1	49	2.0	32	.154	2.46
12	10.0	160	3.8	62	2.5	40	.192	3.08
13	12.0	192	4.6	74	3.0	48	.231	3.69
14	12.0	192	4.6	74	3.0	48	.231	3.69

TABLE V

NORMAL SPAN IN RELATION TO HEIGHT

HEIGHT (Inches)	SPAN		HEIGHT (Inches)	SPAN	
	Male (Inches)	Female (Inches)		Male (Inches)	Female (Inches)
36.0	34.7	34.6	55.0	55.6	54.8
37.0	35.7	35.6	56.0	56.7	55.8
38.0	36.7	36.6	57.0	57.9	56.9
39.0	37.7	37.6	58.0	59.1	58.0
40.0	38.8	38.6	59.0	60.2	59.1
41.0	39.8	39.7	60.0	61.3	60.2
42.0	40.8	40.7	61.0	62.5	61.3
43.0	41.9	41.8	62.0	63.6	62.4
44.0	42.9	42.8	63.0	64.7	63.6
45.0	44.0	43.8	64.0	65.8	64.8
46.0	45.1	44.9	65.0	67.0	66.0
47.0	46.2	46.0	66.0	68.1	67.3
48.0	47.3	47.1	67.0	69.2	68.5
49.0	48.6	48.2	68.0	70.4	69.8
50.0	49.8	49.3	69.0	71.5	71.0
51.0	51.0	50.4	70.0	72.7	72.3
52.0	52.2	51.5	71.0	73.9	73.5
53.0	53.4	52.6	72.0	75.0	74.8
54.0	54.5	53.7			

NORMAL UPPER MEASUREMENT IN RELATION TO HEIGHT

HEIGHT (Inches)	Upper Measurement		HEIGHT (Inches)	Upper Measurement	
	Male (Inches)	Female (Inches)		Male (Inches)	Female (Inches)
36.0	20.9	20.6	55.0	27.4	27.5
37.0	21.3	21.0	56.0	27.8	28.0
38.0	21.7	21.4	57.0	28.3	28.4
39.0	22.1	21.8	58.0	28.7	28.9
40.0	22.4	22.1	59.0	29.1	29.4
41.0	22.7	22.4	60.0	29.6	29.9
42.0	23.1	22.8	61.0	30.0	30.4
43.0	23.4	23.1	62.0	30.5	30.9
44.0	23.7	23.5	63.0	31.0	31.5
45.0	24.0	23.8	64.0	31.5	32.1
46.0	24.3	24.1	65.0	32.0	32.6
47.0	24.6	24.4	66.0	32.5	33.1
48.0	24.9	24.8	67.0	33.1	33.6
49.0	25.2	25.1	68.0	33.7	34.1
50.0	25.6	25.5	69.0	34.3	34.6
51.0	25.9	25.8	70.0	34.8	35.1
52.0	26.3	26.2	71.0	35.2	35.6
53.0	26.7	26.7	72.0	35.6	36.1
54.0	27.0	27.1			

TABLE IV

NORMAL WEIGHTS FOR MEN IN POUNDS (*With Light Clothing and Shoes*)

Age Years	5 ft	5 ft. 2 in.	5 ft. 4 in.	5 ft. 6 in	5 ft. 8 in.	5 ft. 10 in.	6 ft.	6 ft. 2 in.
15	107	112	118	126	134	142	152	162
16	109	114	120	128	136	144	154	164
17	111	116	122	130	138	146	156	166
18	113	118	124	132	140	148	158	168
19	115	120	126	134	142	150	160	170
20	117	122	128	136	144	152	161	171
21	118	123	130	138	145	153	162	172
22	119	124	131	139	146	154	163	173
23	120	125	132	140	147	155	164	175
24	121	126	133	141	148	156	165	177
25	122	126	133	141	149	157	167	179
26	123	127	134	142	150	158	168	180
27	124	128	134	142	151	158	169	181
28	125	129	135	143	152	159	170	182
29–30	126	130	136	144	153	160	172	184
31–33	127	131	137	145	154	162	174	186
34–35	128	132	138	146	155	165	176	188
36–37	129	133	139	147	156	166	178	190
38–39	130	134	140	148	157	167	179	192
40–41	131	135	141	149	158	168	180	193
42–43	132	136	142	150	159	169	181	194
44–45	133	137	143	151	160	170	182	195
46–50	134	138	144	152	161	171	183	197
Over 50	135	139	145	153	163	173	184	198

NORMAL WEIGHTS FOR WOMEN IN POUNDS (*With Light Clothing and Shoes*)

Age Years	4 ft 8 in	4 ft 10 in	5 ft	5 ft 2 in	5 ft 4 in	5 ft. 6 in.	5 ft 8 in	5 ft 10 in.	6 ft
15	101	105	107	112	118	126	134	142	152
16	102	106	109	114	120	128	136	143	153
17	103	107	111	116	122	129	137	144	154
18	104	108	112	117	123	130	138	145	155
19	105	109	113	118	124	131	139	146	155
20	106	110	114	119	125	132	140	147	156
21–22	107	111	115	120	126	133	141	148	157
23	108	112	116	121	127	134	142	150	157
24–25	109	113	117	121	128	135	143	151	158
26–27	110	114	118	122	129	136	144	152	159
28–29	111	115	119	123	130	137	145	153	160
30	112	116	120	124	131	138	146	154	161
31–32	113	117	121	125	132	140	148	155	162
33	114	118	122	126	133	141	149	156	162
34–35	115	119	123	127	134	142	150	157	163
36–37	116	120	124	128	136	143	151	158	164
38	117	121	125	130	137	145	153	160	166
39	118	122	126	131	138	146	154	161	167
40	119	123	127	132	138	146	154	161	167
41–42	120	124	128	133	139	147	155	162	168
43	121	125	129	134	140	148	156	163	170
44–45	122	126	130	135	141	149	157	164	171
46–47	123	127	131	136	142	150	158	165	173
48–49	124	128	132	137	143	152	160	167	175
Over 50	125	129	133	138	144	152	162	170	177

Every patient should be weighed and the weight compared with the usually accepted standard for a person of the same sex, height and age. If coming within ten per cent of the standard he may be regarded as normal, providing no apparent cause can be found for his underweight.

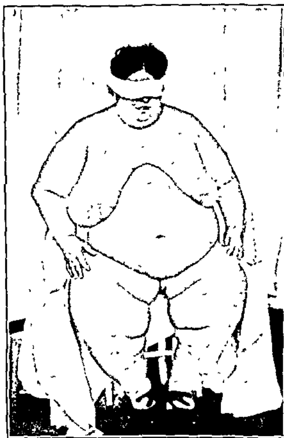


Fig 2—Thyropituitary obesity Note the fat upon the shoulders, breasts and thighs (Philadelphia General Hospital)

Emaciation· This may be the result of malnutrition, wasting diseases or disease of the gastrointestinal canal. Rapid emaciation is a prominent symptom in marasmus, tuberculosis, Simmonds' disease, Addison's disease, cancer, long-standing diabetes, chronic suppuration, hyperthyroidism, long-continued fevers, chronic diarrhea, or stricture of the esophagus. Pyloric obstruction, or infestation with intestinal parasites will have the same effect. Cabot notes loss of sleep as a frequent cause of emaciation, and the increased metabolism of exophthalmic goiter is often evidenced by rapid loss of weight. It is also noted in people who attempt to reduce their weight by starvation and the use of certain drugs.

Obesity. This is often found in apparently normal individuals, particularly in "hearty eaters." On the other hand, obesity is frequently a family or even a racial predisposition and seems to have no relation to the amount of food in-

been arrested by some wasting disease contracted during early childhood, or again, it may be due to some pathological process, such as spinal caries. Also, an unusually tall individual should make one think of endocrine imbalance.

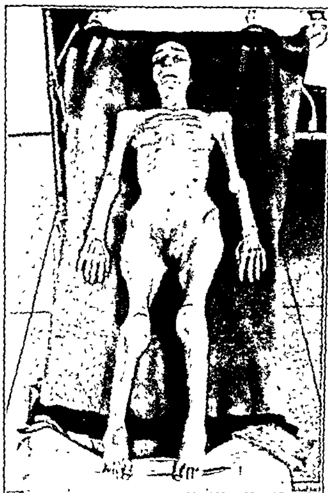


Fig. 1—Simmonds' disease Pituitary cachexia (Courtesy, Dr. L. G Rowntree, Philadelphia General Hospital.)

Muscular Development. This is often governed by the amount of physical exertion to which the individual has been subjected. A patient's muscular development may have formerly been good, but may have become "flabby" because of prolonged febrile disease, chronic diseases, such as tuberculosis or cancer, malnutrition, disease of the digestive apparatus, or some mental disturbance reflexly producing digestive disturbance. Lack of exercise from any cause may also be responsible for a general loss of muscular tone. Asymmetric muscular hypertrophy or atrophy should suggest disease of the central nervous system.

Weight. When an apparently undernourished individual first presents himself for examination, the physician should determine whether he has ever been stouter or if his present state of nutrition is apparently normal for him.

gested. It is sometimes noted that very fat people consume less food than thin ones living under the same conditions, nevertheless, the individual consumes more food than he requires Pathologic obesity may be caused by deficiency of some of the ductless gland secretions, by diminished oxidation, lack of exercise and deprivation of sunshine.

In general, the various parts of the body should be compared to the general stature; if any one member is undersized or overdeveloped, the cause for this condition should, if possible, be ascertained.

Posture and Position: In health, persons will assume certain postures because of muscular development, obesity, training, habit and convenience. During a physical or gynecological examination, patients may be instructed to place themselves in certain definite positions in order to facilitate the examination. The commonest positions utilized for this purpose are as follows:

Sims' Position: (*a*) Anterior and (*b*) posterior view, (*c*) dorsal recumbent position, (*d*) same with leg holders, (*e*) knee-chest position, (*f*) Fowler's position, (*g*) Trendelenburg position, (*h*) Walcher position, (*i*) Edebohl's dorsal position The accompanying illustrations on the opposite page are descriptive of these positions

In certain diseases the patient will assume a definite posture. This does not include chronic bone affections which give rise to deformities, for in these the victim does not assume the posture; rather he has it thrust upon him Definite positions are often assumed in order to relax muscular spasm. Thus, a person who has a spasm in his calf muscles will usually flex his knee. In abdominal muscle spasm both knees are usually flexed, so as to relax the abdominal muscles. When the patient lies upon his back, he may assume this posture voluntarily and it may indicate nothing more than slight illness, unattended by pain

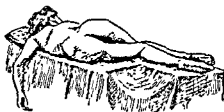


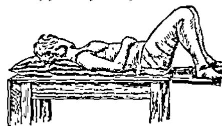
Fig 4—Thorough relaxation (dorsal inertia)

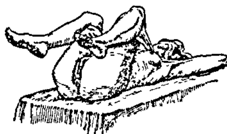
Dorsal Inertia This is a passive posture; the patient lies upon his back, but has a tendency to slip toward the foot of the bed, or perhaps to either side. This is usually noted in conditions of great weakness, most frequently in acute infectious disease, particularly typhoid fever. It is indicative of great muscular weakness and mental apathy. (SEE: Fig. 4.)

Rigid Dorsal Posture: In this posture both legs are drawn up in order to diminish abdominal tension. This is seen, as a rule, in general peritonitis, pelvic peri-


(*a*) Sims' position, anterior view

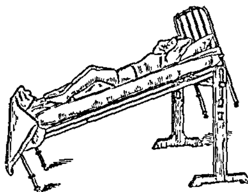

(*b*) Sims' position, posterior view

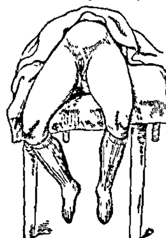

(*c*) Dorsal recumbent position


(*d*) Dorsosacral position, with leg-holder applied


(*e*) Knee-chest, or genupectoral, position


(*g*) Trendelenburg position (*Ashton*)


(*f*) Fowler's position (*Macfarlane*)


(*h*) Walcher position. (*Hirst*)

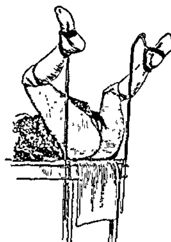

(*i*) Edebohl's dorsal position

Fig. 3—Positions (From *Dorland's Dictionary*)

Pleurosthotonos: The body is arched and in a lateral position usually because of some spinal affection or acute pleural involvement.

Orthotonos: The trunk and the neck are rigidly extended in a straight line; this position is at times seen in strychnia poisoning, tetanus, meningitis or rabies deformity, new growths and comparative lengths; the size, shape and symmetry of the joints should also be noted, and they should be examined as to mobility, tenderness, discoloration and pain.

Any detailed examination of the *bones* must be carried out by the aid of x-rays. By inspection and palpation only such

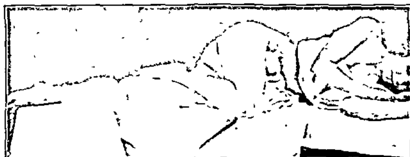


Fig 6—Unilateral posture (subdiaphragmatic abscess; right leg flexed so as to relieve abdominal tension and thoracic pressure).

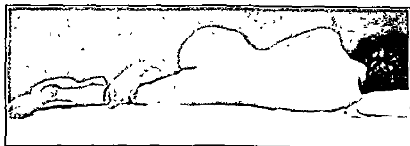


Fig 7—Unilateral posture, acute splenitis, left lower extremity drawn up to relieve left-sided abdominal tension

abnormalities as of contour, exostoses, beading of ribs, craniotabes, saber shins, or fractures of the long bones may be detected. Physical examination of the *joints* is more satisfactory, as palpation will reveal pain or tenderness in the joint or its immediate vicinity; also irregularity in shape, such as the protrusion of the joint-pocket and the filling of its natural depression which is characteristic of effusion. Attachment to the bone, as osteophytes ("lipping") or gouty tophi, which are not attached to the bone, may

A Semireclining Posture: This may be assumed in conditions where there is interference with respiration, particularly disease of the heart after failure of compensation, pleural effusions and asthma. The back is usually supported in order to favor the accessory muscles of respiration. This position is also assumed by convalescent patients who are permitted to sit up gradually before they are allowed to get out of bed.

Bones and Joints: The condition of the long bones should be observed as to

tonitis, at times in meningitis, and in great distention of the abdomen due to ascites or tympanites. *In acute appendicitis,* the right leg is usually drawn up, and this is true also in incarcerated right inguinal hernia, inflammation of the right spermatic cord, right-sided pelvic inflammation or peritonitis, psoas abscess and, at times, when a renal calculus is passing down the right ureter (SEE: Fig. 5) In left-sided local peritonitis or pelvic suppuration, left-sided incarcerated inguinal hernia, acute diverticulitis, psoas abscess or passage of a left ureteral calculus, the left leg will be drawn up.

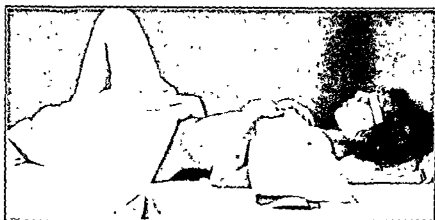


Fig 5—Posture denoting pain in right lower abdomen (acute appendicitis, etc).

Unilateral Posture: The patient will lie on the right side in cases of acute right-sided pleurisy, right-sided lobar pneumonia, or in the presence of a much enlarged liver. This position is assumed in order to support the affected side and limit its movements. He will lie on the left side in cases of left-sided pleurisy, lobar pneumonia, large pericardial effusions, and large left-sided pleural effusions (SEE: Figs. 6 and 7).

Coiled Posture: The patient lies upon one side with the legs drawn up until they meet the trunk. This is noted in meningeal diseases, hepatic, renal, and intestinal colic (SEE: Fig. 8). The *knee-chest position* may be assumed because of some painful condition of the spine or ribs, tumor, or skin lesion of the back.

Prone Posture: This is often assumed for the sake of rest, especially after abdominal pain or colic. Very often this position may be taken because of eroded vertebrae, tuberculosis of the spine, or, at times, to relieve the pain of gastric ulcer, or other severe abdominal colic.

Opisthotonos. This is an uncommon dorsal posture in which the body rests upon the head and heels, the trunk being arched upward. It is noted in strychnia poisoning, tetanus, convulsions of rabies, hysteria, epilepsy and, to a mild degree, in meningitis where the retraction of the head with rigidity of the neck causes the back of the head to bore into the pillow (SEE: Fig. 9).

Emprosthotonos: This posture is the reverse of opisthotonos; the patient's upcurved body rests upon the forehead and feet, face downward. This position is very rarely seen in tetanus and strychnia poisoning.

down upon the heel. This is observed in peripheral neuritis, diabetic neuritis, chronic arsenical poisoning, alcoholism.

Festinating Gait· The whole body is bent forward and is held rigidly; the patient walks upon his toes, having the appearance of being pushed from behind. He starts out slowly, but gradually increases the rapidity of his gait until he is stopped by some object, because he is unable to stop himself. This is noted in paralysis agitans and, at times, as a postencephalitic sequela

feet wide apart, staggers, sways to and fro, often reeling, and adopting a zig-zag course. This may be the result of the presence of a tumor in the cerebellum.

Flat-footed Gait The patient walks with his toes everted, the foot as a whole being placed spade fashion upon the floor; the legs are often slightly bowed.

For a careful examination, the legs and feet should always be bared because the gait may be altered by the presence of local deformities of the knee, hip or ankle joints Very often corns or callosities



Fig 9—Opisthotonos, patient resting on heels and occiput.

upon the toes, heels or, indeed, any part of the foot, due usually to tight or ill-fitting shoes, will cause a limping or abnormal gait. Painful conditions, like erythromelalgia, gangrene, ingrown toenail, or any local inflammatory condition, will alter the normal gait. Speaking generally, the gait is slovenly in persons who are apathetic, weak or anemic, and in those suffering from chronic mental or physical defects; it is hurried in highstrung, nervous individuals. Disease or deformities of the spinal column often cause limping, waddling or other abnormal gaits.

Waddling Gait The shoulders are thrown back, the belly forward, the legs are separated and the patient swings from side to side. This gait is seen in pseudohypertrophic muscular paralysis A similar manner of walking is noted in congenital hip dislocations, and also, at times, in short, obese women during the latter part of pregnancy.

Limping Gait: One foot or leg is dragged; this is due to wasting of the muscles of the affected foot and is seen as a result of infantile paralysis, hemiplegia, monoplegia or paraplegia. Limping may also be due to a painful condition of the bones, as in many forms of arthritis.

Cerebellar Ataxic Gait: This gait resembles that of a person under alcoholic intoxication. The patient walks with his

Local Examination

After the general examination a more detailed *local examination* is begun and the following points should be considered:

be seen or felt. Enlargement or thickening of the capsule, fluctuation (indicative of fluid in the joint), the presence of a palpable "boggy" infiltration and malpositions or distortions of the joints may be palpated in order to ascertain whether they are due to luxations, exudations, necrosis, or pathologic contraction of the muscles.

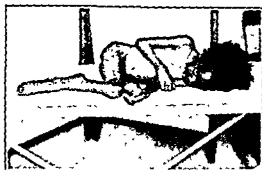


Fig 8—Coiled position (Cerebral pressure with meningeal irritation)

Limitation of motion in a joint may be due to ankylosis, to muscular spasm, to obstruction by the bony growths already mentioned, to adhesion or thickening of the capsular or periarthritic structures, or to pain and effusion of fluid into the joint. In eliciting limitation of motion, comparison with the normal joint is of utmost value.

The detection of a sinus at or near a joint is important, as it indicates the presence of bone necrosis or abscess, or possibly, broken-down gouty tophi.

Reflexes (See: p 831).

The elicitation of reflexes depends upon the patient's general condition, as in very ill subjects many of them must of necessity be omitted Those most commonly tested are the patellar (knee-jerk), tendo Achillis (foot flexion), biceps and triceps, plantar (contraction of the toes), abdominal, and the cremasteric.

In connection with the reflex tests, the examiner should also note muscular efficiency, general tactile sensibility, and ability and manner of locomotion, station and gait.

Gait: The normal gait of different persons varies within wide limits. Watching the feet of thousands of pedestrians, one may observe something peculiar, or at least individual, about each of them There are, however, certain gaits which have come to be regarded as pathognomonic and are seen in local abnormal conditions of the lower extremities and spine, in certain systemic diseases and in various nervous affections. In studying a pathologic gait, one should observe not only the mode of walking, but also the position of the body, the swing of the arms, and the poise of the head

Ataxic Gait. The foot is raised high, thrown forward and suddenly brought down, so that the entire sole of the foot comes in contact with the floor at one time. The body is usually bent forward and the eyes fixed upon the ground. This gait is observed in tabes dorsalis (locomotor ataxia).

Spastic Gait The movements are stiff, the hips and knee joints slightly flexed, the knees seeming to interfere with each other. This gait is seen in spastic paraplegia; it is significant of sclerosis of the lateral pyramidal columns of the cord It may be seen in spinal cord tumor and arachnoiditis. In hemiplegia, the entire leg seems to be thrown out and describes a semicircle before it comes down to the ground.

Paralytic Gait· The feet move very slowly and are dragged upon the floor; the patient stumbles easily. This is seen in chronic myelitis.

Steppage Gait· The patient raises the foot high, turns up the toe and comes

tactile and friction fremitus; by *percussion* to elicit resonance or its modifications and to outline internal organs; and by *auscultation* to determine the quality of breath sounds, voice sound and the presence of adventitious sounds

The Heart: This is examined by *inspection* in order to note the precordium, the location and character of the apex beat and the presence of abnormal areas of pulsation; by *palpation* to determine the site and character of the apex beat and the point of maximum impulse, abnormal pulsations and thrills; by *percussion* for the borders of the heart and for changes of the position of the heart when the patient's position is altered; by *auscultation* for the character of the heart sounds, point of maximum intensity, effect of exertion and of change of posture, rate and rhythm of the heart and murmurs. In connection with the examination of the heart one should take the pulse, noting the rate, rhythm, force, quality and symmetry of the two sides. The blood pressure should also be ascertained with the sphygmomanometer. In some cases an electrocardiographic study becomes necessary.

Abdomen: The abdomen as a whole is examined by *inspection* for size, shape and symmetry, respiratory and peristaltic movements and pulsations, the character of the skin, distribution of hair and the presence of rashes, scars and pigmentation; by *palpation* for muscular rigidity, tenderness, fluctuation and for the size, shape and mobility of the intraabdominal organs and for the presence of tumors and pulsations; by *percussion* for tympany, dullness, flatness, size and position of the organs and for shifting dullness; by *auscultation* for borborygma, hydatid cysts, and in the pregnant uterus for fetal heart sounds; by *auscultatory percussion* for the position and size of the intraäbdominal organs The viscera, *i.e.*, the liver, gallbladder, spleen and kidneys, are examined by *palpation* and *percussion* in order to determine their size, shape, position and the presence of tenderness and fluctuation. The pancreatic region may be palpated for tenderness The intestines are examined by inspection, palpation, percussion and auscultation for distention, tenderness, rigidity, mobility, and borborygma. The distended bladder may be palpated and should be differentiated from a pelvic tumor or enlarged uterus.

Nervous System: The nervous system is examined by *conversation* as to mental process, perversion and mental disturbance; by *inspection* as to palsies, twitchings, station, gait, and general behavior; by *palpation* for tremors, muscle development, abnormal sensations (parasthesia and anesthesia) and sensitive points; by *percussion* for hypersensitiveness and elicitation of reflexes.

Genitourinary System: The bladder should be examined for possible distention, and the urethra for discharge. The external genitals should be examined for scars or abrasions. The condition of the prostate should be noted in the male, and a gynecologic examination made in the female. Inguinal glands and hernial orifices should be palpated.

Back: The spinal column is examined for deformities, as scoliosis, lordosis or kyphosis, for evidence of disease of the individual vertebrae and for limitation of motion anteriorly, posteriorly and laterally. The sacroiliac and lumbosacral areas are to be carefully examined

Head: The head is examined as to size, shape and symmetry; marks of injury; the condition, color and texture of the hair; the position of the head; and the presence of any involuntary movements.

Face: The general expression of the face is observed for signs of stupidity, intelligence, apathy, evidence of suffering, etc, and its size is compared with the rest of the body and especially with the head. The condition of the muscles of expression and mastication and the state of the parotid, submaxillary and other glands are also noted

Eyes: The eyes are examined as to acuteness of vision, limitation of the visual fields; the presence or absence of discoloration or edema of eyelids; ptosis or tremors It is important to note also the color and degree of moisture of the conjunctivae and the presence or absence of petechiae; the equality of the pupils, and their reaction to light and accommodation, the color and mobility of irides; the presence or absence of arcus senilis; as well as the movements of the eyeballs, and whether or not they protrude (exophthalmos) or intrude (enophthalmos).

Nose: The size, shape, color and any evidence of injury are noted as well as the condition of the alae nasi and whether there is any interference with respiration or the presence of discharge One should observe whether the septum is deflected or perforated, the turbinates enlarged or any neoplasm visible and also whether there is any tenderness over the frontal or maxillary sinuses.

Ears: Hearing tests should be applied, the simplest one being the watch test The color and size of the external ear should be observed, especial care taken to detect any tophi; the presence of any discharge from the middle or inner ear has diagnostic importance, also any tenderness in front or back of the ear upon pressure; the drum should be examined for inflammation, bulging, perforation, scars, or the presence of any anomaly.

Mouth: Observe the color, size and degree of moisture of the lips; any asymmetry of the angles of the mouth; any rashes or abrasions, fissures or crusts; areas of discoloration as in Addison's disease; the general hygienic state of the mouth and the odor of the breath.

Teeth: The general condition of the teeth and gums, loose or missing teeth, caries of the teeth, presence of roots, or broken teeth, characteristics of teeth, *i. e.*, Hutchinson's teeth, rachitic teeth, etc., are to be noted.

Tongue: Note its size and the manner in which the patient protrudes it; also whether it is clean or coated, and if any scars or abrasions are visible upon it Also examine it for tremors, color, fissures and any rash which may be observable upon it.

Pharynx and Larynx: These are examined as to color; the condition of the tonsils, if hypertrophied or giving evidence of abscess; the color of the anterior pillars; the condition of the uvula, larynx, arytenoids and vocal cords; the presence or absence of cough and its character; phonation and its character

Neck: Note the general dimensions and any enlargement of the thyroid or other glands; also the presence of any pulsations, arterial and venous; any tracheal tugging or tracheal deviation from midline.

Thorax: Lungs: The lungs are examined by *inspection* for the type of chest, respiratory expansion and visible abnormalities; by *palpation* for the confirmation of inspected signs and to elicit

SECTION 4

Skin and Mucous Membranes

CHAPTER VII

Examination and Diseases of the Skin and Mucous Membranes

The Skin

The skin is examined for:

I. Color.
II. Rashes.
III. Scars.
IV. Temperature.
V. Edema.
VI. Moisture.

I. Color

The complexion of the skin among light skinned people depends largely upon the amount of distention or fullness of the capillaries supplying it. The complexion is also altered by exposure to the sun's rays, to high winds, and to a combination of sun, wind and air.

A. **Tanning**: Tanned, rather hardened skin is common in laborers who are employed outdoors, in drivers, sailors and in others who continually expose themselves to the elements, strong sunlight and artificial rays.

B **Pallor**: Habitual pallor is noted in persons who lead an indoor life, and is seen particularly among prisoners and night workers who sleep during the day.

Pallor is produced by the following conditions: 1. A diminution of the volume of circulating blood. 2. A decrease in the number of red blood corpuscles 3 Failure of the capillaries to fill completely.

Pallor may come on gradually or suddenly; and may be transient or constant. Continuous pallor is noted in all forms of anemia, primary and secondary. Evanescent pallor is often seen in cases of temporary heart weakness, as in syncope, chills and rigors, shock and certain vasomotor spasms. Sudden but persistent pallor, especially if associated with shock, may be a sign of rapid intense hemorrhage. The pallor encountered in nephritis is often out of proportion to the blood picture and may be due to a superficial anemia.

Pallor is also a prominent symptom in acute poisoning and toxic febrile affections and is in evidence immediately before death. Pallor of gradual development which becomes permanent, is either an indication of primary anemia, that is disease of the blood-making organs, or of secondary anemia as in wasting diseases.

The primary anemias are represented by pernicious anemia and chlorosis; and the secondary anemias are seen in: Cancer; arsenical poisoning; chronic febrile disease; chronic gastrointestinal disease; chronic suppuration; chronic mercurial poisoning; chronic lead poisoning; after hemorrhages, *e.g.*, from hemorrhoids, epistaxis, hemoptysis, hematemesis, etc.; leukemia; cachexia; myxedema; nephritis; nephrosis; certain parasitic diseases, *e.g.*, tapeworm, uncinariasis, etc.; syphilis; tuberculosis, and chronic malaria.

Changes of climate may gradually produce a more or less permanent pallor, as in the case of emigrants from a cooler to a warm climate.



pain which may prevent respiration, as in pleurodynia, pleurisy and peritonitis; diseases of the circulatory system, as affections of the heart and arteries, including valvular disease after failure of compensation; congenital stenosis of the pulmonary artery, patulous foramen ovale, disease of the heart muscle (during failure of compensation); large pericardial exudation hindering the heart's action; emphysema and other conditions obstructing the circulation by compressing the capillaries; tuberculosis (later stages); and pressure of mediastinal tumors upon the union of the superior and inferior vena cava at their junction with the right auricle.

Cyanosis may also be caused by overdoses of certain drugs, *e. g.*, antipyrin, acetanilide, opium, hydrocyanic acid, calcium chloride, nitrobenzol, illuminating gas or any other gas causing asphyxiation

Generalized argyria may be mistaken for cyanosis

Erythremia and *polycythemia* are characterized by generalized "erythematous cyanosis," and, as the names imply, by an excessive number of red corpuscles in the circulation

Local venous stasis is caused by compression or obliteration of one of the large venous trunks, the stasis being confined to the region drained by that vessel Thus, pressure of a tumor or aneurysm upon the jugular, subclavian, innominate or inferior vena cava will produce cyanosis of the head, neck and upper extremity, corresponding to the point of pressure. Pressure caused by ascites, tumors and effusions in the peritoneal cavity, or thrombosis of the iliac veins will produce cyanosis of the lower extremities Vasomotor derangements may cause cyanosis, and it may also be produced by cold, or paralysis of certain parts of the body, and by sluggishness or partial obstruction of the circulation and by disease of an artery or vein.

E. Jaundice: Jaundice (icterus) is a term applied to a yellowish coloration of the skin, mucous and serous membranes, and the liquid secretions and excretions of the body. The degree of coloration of the skin varies from a slight yellow tinge to a deep greenish yellow or even an olive green, depending upon the amount of bile pigment present in the circulating blood. In longstanding severe cases the skin assumes a dark, yellowish brown or blackish color as a result of degenerative changes. The skin should, whenever possible, be examined in daylight or under a white light, as ordinary artificial illumination will mask even a moderate degree of jaundice. When in doubt as to the existence of jaundice it may be made more apparent by stretching the skin of the palm of the hand, or by pressing upon the skin, or upon the mucous membrane of the everted lip with a glass slide through which the yellowish color may be seen. Bile pigments are also present in the urine, sweat and sometimes in the milk, salivary secretions and tears.

Jaundice is a symptom found in several diseased conditions, and is not a distinct entity. It may be found in any condition that will obstruct the biliary passages or ducts so as to cause retention of bile in the liver; also in conditions which cause blood destruction, disease of the liver cells and the circulation of certain toxins in the blood.

There are three general types of jaundice though they cannot always be isolated Two or all three types may occur in the same individual at the same time.

C. **Redness:** *General congestion or* hyperemia of the cutaneous capillaries produces this condition; it may be general or local.

General redness is seen in plethoric individuals and, pathologically, in cases of acute fever, especially if continuous, in certain eruptive diseases and in polycythemia. It may also be produced by drugs, *e. g.*, atropine poisoning, alcoholism, etc.

Local Redness: The skin of the face and of the exposed portions of the body appear more red in those who are exposed *to sunlight, open air and mountain* climate than in those who are confined indoors and at low altitudes. Local redness may also be noted in chronic alcoholism, particularly if associated with portal obstruction; in certain vasomotor disturbances, pyrexia, and, at times, in tuberculosis ("hectic flush"), also in chlorosis florida or chlorosis rubra. One-sided redness of the face may be seen on the affected side in lobar pneumonia Local redness, associated with pain, is seen in all local inflammatory conditions and in erythromelalgia (Weir Mitchell's disease).

D. **Cyanosis:** This condition, which varies from a slight bluish tint to a dark *purple discoloration, is dependent upon* the presence of venous blood in the capillaries. It is best observed in the lips, mucous membranes, finger tips and external ear because of the thinness and translucency of their epithelial covering. Extreme cyanosis is noted over the entire body as a dusky leaden tint.

Cyanosis, whether general or local, is always an indication of a deficiency of oxygen and an excess of carbon dioxide in the blood; hence, it is observed in conditions marked by disturbance of respiration and general circulation. *Deficient oxygenation of the blood* occurs when not enough pure air enters the lungs to oxygenate the blood, or when not enough blood is brought in contact with the air in the lungs to promote efficient oxygenation Again, the venous blood in a given area may be unable to circulate at a sufficiently rapid rate to cause proper interchange

Cyanosis may be caused by pathologic conditions interfering with the entrance of air into *the respiratory tract, such* as inflammation of the pharynx and larynx, retropharyngeal abscess; angina *Ludovici; edema of the glottis;* spasmodic croup; laryngeal diphtheria; tuberculous and syphilitic inflammation of the larynx; diphtheritic inflammation of the larynx, trachea and bronchi; obstruction by foreign bodies (pins, food, etc.); tumors of the larynx and upper air passages, paralysis of the dilators of the larynx; pressure by mediastinal tumors such as goiter, aortic or subclavian aneurysm, enlarged bronchial glands, etc.; also because of enlarged thymus; severe diffuse bronchitis; bronchial asthma; whooping cough during a paroxysm, and convulsions Other causes for cyanosis are affections which hinder lung expansion, such as emphy*sema; all forms of consolidation of the* lungs; paralysis of the muscles of respiration; peritonitis (by causing paraly*sis of the diaphragm); pleuritis and* large pericardial exudation; pneumothorax; hydrothorax; hydropneumothorax and pyopneumothorax; *pul*monary edema; tumors of the chest cavity; tumors of the abdomen, pressing upward; epilepsy (during the attack); strychnine poisoning; tetany by causing respiratory spasm; progressive muscular dystrophy; trichinosis; myasthenia gravis; myositis ossificans;

II. Rashes

Rashes or exanthemata are eruptive lesions resulting from pathologic processes in the skin and are usually classified into primary and secondary.

Primary Lesions: The primary lesion represents the pathologic process up to the acme of its development. The following skin lesions belong in the primary classification.

Macules: Spots of various sizes, shapes and colors visible on the skin, which are neither elevated nor depressed.

Vesicles (*Blisters*): Hemp seed to lentil-sized, rounded, acuminated, transparent, opaque or dark elevations of the epidermis, filled with serous, seropurulent or bloody fluid

Bullae or Blebs (*Large blisters*): Irregularly shaped elevations of the epidermis, varying in size from that of a bean to that of a goose egg, and containing serous or seropurulent fluid.

Pustules: Circumscribed, rounded, flat, acuminated or umbilicated elevations of the epidermis containing pus.

Papules (*Pimples*): Millet seed to lentil-sized, circumscribed, solid, elevated, pathologic formations.

Tubercles (*Nodules*): Circumscribed, firm, rounded or acuminated, deeply seated or elevated formations in the skin, varying from the size of a pea to that of a hazel nut.

Wheals or Pomphi: Round, oval or elongated, firm elevations of the skin, pale or slightly reddish in color; are evanescent and cause itching.

Tumors: Hard elevations of tissue varying in size from a hazel nut upwards.

Secondary Lesions: These are the result of primary lesions and are known as:

Crusts: Masses of dried serous or seropurulent exudations on the free surface.

Excoriations: Areas of loss of epidermis because of trauma or the action of chemical agents.

Fissures: Linear breaks in the continuity of the epidermis.

Pigmentations: Areas of increased pigment or color in the skin in consequence of chronic inflammation, new growth formation or trophic disturbance, either temporary or permanent.

Scales: Thin, dry, plate-like flakes compacted and shed from the cutaneous surface.

Scars: Reddish, brownish or whitish new formations of connective tissue, occupying the place of lost normal tissue.

Ulcers: Irregularly-sized and shaped excavations in the skin, the result of suppurative processes.

Secondary lesions either are the result of healed or healing primary lesions or are destructive remnants of primary lesions. These are (1) crusts, (2) scales and (3) ulcers

Primary Lesions

1. **Macules:** The various macules are:

(*a*) ***Hyperemia:*** Bright red areas, which disappear upon pressure.

(*b*) ***Roseola:*** Reddened spots, varying in size from that of a lentil to that of the fingernail.

(*c*) ***Erythemia:*** Diffused redness over a considerable area.

(*d*) ***Telangiectasis:*** Acquired hyperemic spots which can be seen to include large blood vessels

(*e*) ***Nevi Vasculosi:*** Hyperemic spots due to hypertrophy of the capillaries containing visible blood vessels.

as is often indicated by the van den Bergh test. They are generally classified as follows:

I. Obstructive or Hepatogenous Jaundice.
II. Hemolytic Jaundice
III. Suppression Jaundice (Infectious Hepatic; Toxic). (SEE 601.)

F. **Other Discolorations:**

Yellowish, brownish or blackish diffuse patches particularly on the face are seen in chloasma (so-called liver spots).

Yellowish-brown or fawn colored macules associated with larger coalesced areas and covered with furfuraceous scales over the covered portions of the body are characteristic of Tinea Versicolor

Brown indurated areas of skin which are dry, smooth and glossy are found in scleroderma.

Dark brown to bluish black discoloration of the entire skin surface is seen in hemochromatosis. This is associated with liver enlargement and hyperglycemia (bronze diabetes).

Dirty yellow to deep brown pigmented areas in the axillae, under the breasts, in the inguinal regions, over the abdomen, and in the flexor folds which are associated with papillary thickening of the skin are found in acanthosis nigricans. This may occur in abdominal malignancy.

Dark brown, gray or black pigmentations of the face, hands, feet and the knuckles and tendons of the hands and feet associated with dark colored urine (alcaptonuria) are found in ochronosis.

Dark pigmented areas or nodules which have a tendency to coalesce are seen in melanotic malignancy

Bronzing of the skin may be seen in Hodgkin's disease. It is also found in many cases of Addison's disease. The color of the skin ranges from light yellow to deep brown or black slate color. It is more marked in those portions of the body which normally contain pigment, such as the face and hands, and around the waist line; it is also seen upon the mucous membrane; the fingernails and cornea usually remain clear. Very dark areas of discoloration may be seen early on the palate and near the anus.

Local bronzing may be caused by certain dyes or metals; continuous exposure to the sun; and it also occurs in the early stages of pellagra.

Arseno-melanosis is a form of bronzing which sometimes discolors the skin and mucous membrane of the mouth after the prolonged administration of arsenic; it is often seen on the palms of the hands and usually disappears when the drug is discontinued.

Gray skin (argyria) is a grayish discoloration of the skin caused by the long continued internal administration of silver salts It consists of a deposit of small granular patches of metallic silver, or of silver compounds, in the skin. The discoloration is bluish gray, more marked upon the hands and face, it is not altered by pressure. The discoloration is also observed in the mucous membrane of the mouth and in the serous membranes and in the internal organs.

Carotinemia causes a yellowish discoloration of the skin due to the ingestion of carrots or other yellow pigmented vegetables. The palms and soles are deepest stained The bilirubin in the blood is normal.

pains, are the diagnostic features. The lesions may appear as separate rings (*erythema annulare*); as concentric rings (*erythema iris*); in disc-shaped patches with elevated edges (*erythema marginatum*); or in a variously figured arrangement (*erythema figuratum*); or variously distributed red elevations (*erythema nodosum*).

Pellagra· Pellagra is an endemic remittent deficiency disease due to imbalanced protein-poor diet, lacking in vitamins B_2 and B_6. These substances are found in large quantities in brewer's yeast, in liver and other foods. Pellagra is found more often in institutions and among alcoholics, and is more prevalent in the spring and autumn than at other seasons.

Pellagra is characterized by gastrointestinal symptoms, nervous disturbances and characteristic skin lesions. The lesions are found upon the back of the hands, face, neck and dorsal surface of the feet (the parts exposed to the sun). The lesions are at first erythematous and gradually become darker, the skin often desquamates or vesicles and bullae evacuate, leaving a dry, deeply stained and fissured surface of a mahogany red color (SEE· Fig 3, p 134).

Pityriasis Rosea This eruption is found on the trunk, appearing obliquely to the ribs The lesions are of rose red color and slightly scaly, having a central clearing. The scales are dry. "The primitive patch or sentinel spot" is a characteristic finding Subjective phenomena are usually absent.

Pediculosis Corporis The bites of lice may produce a minute red or purple eruption The small size of the lesions, their confinement to the covered parts, the intense itching with evidence of scratch marks, and the discovery of pediculi or nits on the clothes, are the diagnostic features.

Measles (*Morbilli, Rubeola*): Preceding the rash there is fever, lacrimation and coryza. The rash appears first upon the face as small red spots, and later spreads over the entire body as dusky red macules arranged in crescentic patches



Fig 2—Erythema multiforme.

Rubella, Rötheln (German measles): This affection produces a macular or maculopapular rash which disappears by slight desquamation in two or three days. The moderate fever, sore throat, swollen cervical glands, and history of contagion will assist in the diagnosis.

Accidental Rashes. Local inflammation like tonsillitis and acute gastritis,

(*f*) ***Areola:*** A hyperemic area surrounding a skin lesion, *e.g.*, the area surrounding a boil

(*g*) ***Purpura:*** Small hemorrhagic spots which do not disappear upon pressure.

(*h*) ***Petechiae:*** Hemorrhagic spots the size of a pin point.

(*i*) ***Vibices:*** Long narrow streaklike hemorrhagic lesions; due to a linear subcutaneous effusion of blood.

(*j*) ***Ecchymosis:*** Large irregularly-shaped hemorrhagic areas. The red color usually gives way to blue, greenish brown or yellow after a definite time has elapsed.

(*k*) ***Achromia:*** Hereditary circumscribed areas which are deficient in pigment.

(*l*) ***Albinism:*** Large generalized areas deficient in pigment.

(*m*) ***Vitiligo:*** Acquired areas of deficiency in pigmentation.

(*n*) ***Chloasma:*** Yellowish brown spots frequently seen on the faces of women who have borne children, or who suffer from uterine diseases.

(*o*) ***Lentigines*** (freckles): Groups of yellowish brown pigmented spots

(*p*) ***Nevi Pigmentosi and Nevi Spili, etc.:*** Congenital pigmented spots in the skin, varying in color from light brown to almost black; nevus spilus is characterized by a smooth surface.

(*q*) ***Discoloration:*** A change in the color of a large part of the body. This condition is met with in icterus, chlorosis, leprosy, malignant disease, and staining from the internal administration of nitrate of silver.

A Generalized Red Macular Eruption: This is observed in the following conditions:

Syphilis: Secondary syphilis may manifest itself as an eruption of small red macules. They are usually abundant and frequently cover the entire body; subjective symptoms are lacking but they are usually associated with a history of or with evidences of syphilis, such as the scar of a chancre, pain in the bones, alopecia, swollen glands and sore throat.



Fig 1—Secondary syphilis (macular rash)

Erythema Multiforme This may be manifested as a macular eruption, though the macules are usually associated with dark red papules or tubercles The multiformity of the lesions, their preference for the extremities, their appearance in successive crops, the short duration of each lesion, the absence of subjective phenomena, such as itching and burning, and the presence of rheumatic

I

II

THE COMMONER TYPES OF INFECTIOUS EXANTHEMATA

1. Chickenpox (varicella), the type of the vesicular exanthemata.

2. Smallpox (variola), the type of the pustular exanthemata, illustrating clearly the essential eruptive lesion (the pustule).

3. Scarlet fever (scarlatina), the type of the scarlatinoid exanthemata, affording a good illustration of the maximal degree of eruption at the natural skin folds

4. Rubella (German measles), a rare, seasonal, epidemic, contagious disorder characterized "by a general glandular enlargement, itching, and a rash" (Sabouraud)

5. Measles (rubeola), the type of the "morbilliform" rashes

6. Florid measles, almost purpuric in appearance, constituting, from the eruptive standpoint alone, a manifest transitional form between the morbilliform rash (hyperemic) and the purpuric eruption (hemorrhagic);

V

VI

glands, rapid pulse and the punctiform character of the rash will indicate the diagnosis.

Variola (Smallpox). The initial rash is at first macular. The spots are bright red and appear first upon the forehead, back of the wrists and hands, and in the mouth; it then spreads to the face, trunk and extremities, palms and soles. Later these macules turn to papules.

Local Irritation: Local irritation from traumatism, excessive heat or cold, ex-



Fig. 6—Peliosis rheumatica.

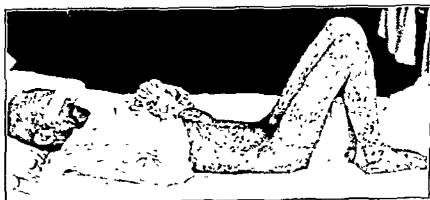
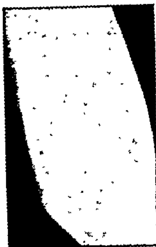


Fig 7—Arsenical dermatitis.

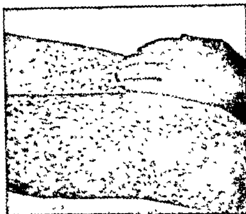

I


II


III


IV


V


VI

The Commoner Types of Infectious Exanthemata

posure to the sun and other light rays, poisonous plants or drugs may produce erythema.

Erythema Intertrigo (chafing): This occurs where two cutaneous surfaces come in contact. The parts are red, moist and sometimes macerated. The condition excites a burning pain



Fig 8—Erysipelas (Doane's case).

Erysipelas In this disease there is at first intense local redness of the skin; it often affects the face and neck; the eruption which begins on the first or second day of the disease consists of dark red, spreading patches of erythema, having a sharp line of demarcation Edema and infiltration of the underlying tissues, cause intense itching and burning. There is high fever and other constitutional symptoms.

Acne Rosacea: This is a chronic disease; the redness appears on the face, particularly the nose and cheeks. It is associated with inflammatory lesions of the sebaceous glands and dilated capillaries. The facial hyperemia, acneform lesions, telangiectasis and the hypertrophy of the skin of the nose (rhinophyma) may remain permanent (See: Fig 9, p. 138).

Brown Macules: These are observed in.

Lentigo or Freckles. The spots are small and found especially on exposed parts—face, neck, shoulders and hands

Chloasma · Dark brown spots may result from irritation of the skin by the action of chemicals, heat, scratches, or blisters. They are sometimes noted in general diseases like Addison's disease and syphilis. They also occur in primary affections of the skin, as vitiligo, morphea, scleroderma and leprosy.

Tinea Versicolor: This is caused by the *Microsporon furfur.* The lesions are fawn-colored macules covered with furfuraceous scales. They appear upon the chest, shoulders, back, neck and upper arm The lesions are at first discrete but soon coalesce.

Moles or Nevus Pigmentosa: These consist of congenital deposits of pigment upon various parts of the body

White or Pale Yellow Macules: These are observed in:

Vitiligo: Apart from the absence of pigment, the skin is normal in appearance and function. An excess of pigment is generally noted at the periphery of the white patches.

Leprosy: In this condition there are structural changes in the skin and anesthesia in addition to the white appear-

brospinal meningitis, typhoid fever, and in the "common cold."

Dermatitis Venenata: A vesicular eruption may result from contact with poisonous plants, such as the poison ivy or oak. The eruption generally appears on the exposed parts—face or hands;

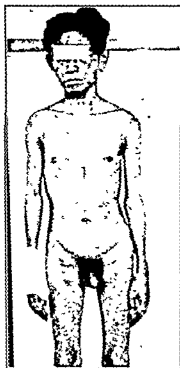


Fig 10—Leprosy

the affected part is red and swollen and there is intense itching

Dermatitis Herpetiformis or Multiformis (Duhring's disease) · The vesicles are irregular in shape, and appear in clusters; are tense, show no tendency to rupture and are frequently associated with other lesions—papules, pustules and bullae. They excite intense itching and burning and appear in successive crops over a period of weeks or months.

Impetigo Contagiosa: The eruption consists of small vesicles which subsequently enlarge and may reach the size of blebs; they appear in crops and are commonly discrete. They are usually flat and unbilicated and are filled with a straw-colored fluid; they show no tendency to break, but dry up so as to form thin yellow crusts which excite but little itching. The disease is contagious and autoinoculable. It occurs especially in children.

Vesicular Eczema: The vesicles are quite small and aggregated in patches; the intervening skin is red and thickened; the vesicles tend to break and pour forth a serous fluid which keeps the part moist. The eruption is associated with intense itching.



Fig 11—Herpes zoster.

Miliaria, or Heat Rash or Prickly Heat: This is an acute inflammation of the sweat glands. They may appear as an eruption of minute vesicles, always discrete and surrounded by red areolae. Their site of preference is the trunk and they are generally associated with pin-

ance. The tubercular form of leprosy presents erythema, pigmentation, tubercles and ulcerations. The lesions are found upon the face, extremities and genitals.

Morphea: In the late stage of this affection, the circumscribed patches are white or yellow. The structure of the skin is altered, and the periphery of the patches is distinctly hyperemic.

Facial Hemiatrophy: The onset of this disease may be marked by the appearance of a yellow or white spot on one side of the face.

2. **Vesicles:** A vesicle or "blister" is a small elevation of the skin, containing serous fluid, and varying from the size of a pinhead to that of a split pea. Vesicles are observed in the following conditions:

Sudamina: This consists of an eruption of minute vesicles which result from the imprisonment of sweat in the layers of the skin. It is usually associated with free perspiration; the vesicles are translucent, lacking inflammatory characteristics, and show no tendency to rupture

Herpes Zoster: The vesicles appear in groups or clusters; they are mounted on an inflammatory base; show no tendency to rupture, and are frequently associated with burning or neuralgic pains. The eruption is distributed along the line of the nerve trunks.

Herpes faciales occurs in many febrile diseases, such as lobar pneumonia, cere-



Fig 9—Acne rosacea.

4. **Pustules:** A pustule is a small circumscribed elevation of the skin containing pus. Pustules are observed in the following diseases:

Eczema Pustulosum The pustules are small and are aggregated in patches. They are generally associated with minute vesicles, the intervening skin being red and thickened; there is marked burning and itching.

Acne Vulgaris The pustules are usually confined to the face, back and shoulders They have their origin in the sebaceous follicles, are generally associated with papules and comedones ("blackheads") and excite no itching.

Sycosis Vulgaris The pustules follow the reddish papules They are pierced by a hair, seldom rupture but form crusts Pustules also occur in glanders, anthrax, sporotrichosis and local skin infections.

Dermatitis Herpetiformis: SEE pp 139 and 140

Impetigo Simplex This affection is usually observed in children, the pustules are round and range in size from a pea to a cherry. There is only a slight red areola and this finally disappears The pustules remain discrete and show little tendency to rupture, but dry up and form yellowish brown crusts They are most frequently observed on the extremities and excite no itching The disease lasts from a few days to a week or longer.

Varicella or Chickenpox The pustules are secondary to vesicles; they appear especially on the trunk and hairy scalp and are small and not umbilicated They are seen in association with vesicles and scabs and excite but little itching. Some fever accompanies the eruption.

Ecthyma This disease is observed especially in poorly nourished adults The pustules vary in size from a pea to a cherry; are few in number, mounted on an inflammatory base, surrounded by a distinct inflammatory areola, and excite but little itching. They seldom break, but dry up and form brownish crusts.



Fig 13—Dermatitis herpetiforma

Smallpox In this disease shot-like papules and umbilicated vesicles precede the pustules. The latter are small, surrounded by a red areola, and usually excite some itching They occur in greatest numbers upon the face and back of the hands. The high fever and history of contagion will assist in making the diagnosis

Syphilis: The pustules are frequently associated with other lesions; they are often mounted on a copper-colored inflammatory base. They excite no itching and can usually be recognized by the history and other evidences of syphilis

head papules which show no tendency to rupture. This rash causes a little burning and itching. The disease is due to excessive sweating and occurs in hot weather.

Scabies: In this affection the vesicles are small and usually associated with pustules and burrows; these excite intense itching. They are found most frequently in the finger webs and forearms, in the axillae, under the mammae, and *on the inner aspect of the thighs.*

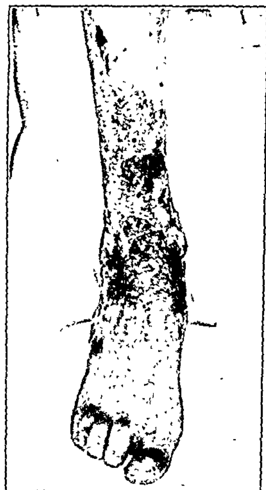


Fig. 12—Pemphigus

Tinea Circinata (ringworm): The vesicles are small, appearing around the margins of a generally circular lesion having a clear center.

Varicella (chickenpox): The papular lesions vesicate and remain firm.

Variola (smallpox): Umbilicated vesicles appear on the fifth or sixth day of the disease.

Syringomyelia: A vesicular rash may occur in certain nerve areas and in analgesic zones The vesicles may last several days, are painless and nonirritating

Miscellaneous Conditions: Vesicles may also occur in anthrax, foot and mouth disease, erythema multiforme, dermatitis repens, dermatitis medicamentosa, etc.

3. **Blebs or Bullae:** A bleb, or bulla, is a circumscribed elevation of the skin, containing serous fluid, and varying in size from that of a pea to an egg. Blebs are observed in the following conditions.

Dermatitis Herpetiformis: The bullae are frequently associated with papules, vesicles and pustules; they are surrounded by inflamed skin and appear in clusters, show no tendency to break, but dry up and leave yellowish brown crusts They excite considerable itching.

Pemphigus: The bullae appear in crops; they itch but little, lack an inflammatory areola, and as a rule dry up, leaving behind a thin pellicle. The disease is generally chronic and usually fatal

Syphilis: The bullous syphilide is observed in hereditary syphilis, and very late in the acquired form of the disease The contents of the bullae soon become pustular, the blebs dry up and form dark green, cone-shaped, stratified crusts, which become detached and leave discharging ulcers. The history and the other evidences of syphilis will aid in the diagnosis.

Yaws (Framboesia, Pian, Parangi, Bubo, Coco): This is a contagious inoculable tropical disease occurring in dark skin natives of South America, parts of Africa and some of the Pacific islands. It is caused by the Treponema pertenue and is of nonvenereal origin. The lesions pass through three stages The primary stage manifests itself, after an incubation period of from two to four weeks, as an extragenital papule which becomes crusted and has a granulating base The second stage is characterized by a generalized eruption of papules which become crusty and have granulating bases These lesions heal slowly and leave pigmented areas. The lymph nodes are enlarged but do not suppurate The tertiary stage shows ulcerative nodular lesions that may involve the skin or the bones, often the nose, pharynx and palate The Wassermann reaction is positive

Prurigo The papules are small, pale and deep seated and are accompanied by intense itching The disease begins in early childhood and lasts throughout life

Lichen Planus The papules are small, angular and of purplish color They are often arranged in rows upon the extensor surfaces of the legs, the flexor surfaces of the arms and occasionally on the trunk, buccal mucous membrane and male genitalia *They cause intense itching.*

Smallpox The papules are hard and have a shot-like feel; they soon terminate in umbilicated vesicles They excite some itching and are associated with high fever, pain in the back, and usually with *a history of contagion.*

Measles The papules are small and run together to form crescent-shaped patches; they are associated with moderate fever, swollen cervical glands, coryza, conjunctivitis, and bronchitis There is often a history of contagion.

6. **Tubercles:** Tubercles are large, circumscribed, solid elevations of the skin varying in size from a large pea to a hazel nut. They are observed in the following conditions:

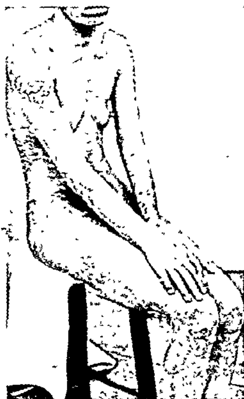


Fig 16—Papular syphiloderm.

Erythema Nodosum: The tubercles are large and usually appear on the extremities. They are reddish purple in color and never suppurate, and are often associated with malaise, fever and rheumatic pains

Erythema Multiforme: The tubercles generally appear in conjunction with macules and papules. They are flat and of a bright red or purple color, appear-

Furunculosis: The deep indurated area becomes localized and forms a red, tender, hot mass which fluctuates and later ruptures.

Drug Eruptions: Drug eruptions as from bromides, iodides, arsenic, copaiba



Fig 14—Pustular secondary syphilis.

and other drugs either taken internally or applied topically may cause various kinds and sizes of pustules.

5. **Papules:** A papule is a circumscribed solid elevation of the skin varying in size from a pinhead to a pea. Papules are observed in the following conditions:

Erythema Multiforme: The papules are often associated with macules and tubercles; they are flat and are of a bright red or purple color. They appear especially on the extremities and show no tendency to suppurate, but gradually disappear in the course of two or three weeks. They excite no itching, but are often accompanied by prostration and rheumatic pains.

After the Use of Certain Drugs: Bromides, iodides, copaiba, cubebs, and coal-tar products may produce a papular eruption. The history will aid in the diagnosis.

Eczema Papulosum: The papules are very small, closely aggregated, and often associated with vesicles and pustules, the skin is thickened and there is intense itching

Syphilis. The papules are dark in color, and widely distributed, being especially marked on the trunk and flexor surfaces of the extremities; they are usually associated with pustules and excite no itching. The history and the accompanying evidences of syphilis will aid materially in establishing the diagnosis.



Fig 15—Yaws
(Philadelphia General Hospital.)

dry, brittle and loose. The microscope will reveal the presence of the *Trichophyton tonsurans*.

Leprosy: One form of leprosy manifests itself with tubercle formation of a pale red or yellow color, which under-



Fig 19—Favus

goes slow absorption or ulceration There is usually more or less anesthesia in the parts affected

7 **Wheals, or Pomphi:** Wheals are evanescent elevations of the skin, generally more or less round, and often white in the center and pale red at the periphery They excite considerable itching. They are observed in the following conditions

Insect Bites The bites of certain insects, such as mosquitoes, bees, beach flies, etc, may cause wheals surrounded by areas of erythema and cause itching.

Urticaria The wheals appear in crops, are of short duration and may appear on any part of the body. They excite intense itching.

Allergy: The urticarial lesions or wheals appear as a result of the ingestion of certain kinds of food, or because of the introduction of a foreign protein into the body.

Angioneurotic Edema· This is characterized by the appearance of evanescent wheals. The deeper structures of the skin are often invaded, causing hard raised areas that may be painful

Secondary Lesions

1. **Crusts:** Crusts consist of dried exudations and may be red, yellow, brown or green in color They are marked in the following diseases.

Eczema The crusts are generally associated with pustules and vesicles,



Fig 20—Tinea tonsurans

the surrounding skin is red and thickened and there is considerable itching.

Seborrhea· The crusts of seborrhea are generally observed on the scalp; itching is absent or only slight, and there are no evidences of inflammation

ing especially on the extremities and showing no tendency to suppurate, but gradually disappearing in the course of two or three weeks. They excite no itching, but are often associated with prostration and rheumatic pain. The dis-



Fig 17—Neurofibromatosis (Von Recklinghausen's disease)

ease is probably allied to erythema nodosum.

Lupus Vulgaris This may begin either as a papule or a tubercle. It is especially observed on the face. The tubercles are pale red and are quite soft to the touch, as a rule slowly breaking down and forming shallow ulcers, with soft red margins The ulcers are painless and secrete but little material. They may invade all of the soft structures, but the bones escape.

Syphilis: The tubercular syphilide manifests itself as dark red tubercles. There are seldom more than three or four at a time and they generally appear on the face and extremities. They are firm and often break down, forming deep, punched-out ulcers which secrete an abundant purulent material.

Keloid (scar tissue): This is characterized by elevated smooth, irregularly shaped whitish or pinkish ridges, nodules or plates of dense connective tissue. usually as a result of traumatism Keloids are commonest in the colored races.

Neurofibromatosis (Von Recklinghausen's disease): The tumors may be large or small and may occur in large numbers upon the trunk and extremities, they are not tender to touch, and do not burn or itch. Areas of pigmentation



Fig 18—Tinea sycosis.

are often distributed over the surface of unaffected skin.

Tinea Sycosis, or Barber's Itch: The tubercles appear on the hairy parts of the face and involve the hair follicles Suppuration soon begins in the center of the tubercles, and the hairs become

Ichthyosis: This affection is either congenital or begins in early life The scales are dry, and are especially marked on the extensor surfaces, face, trunk and abdomen. Itching is absent, and there is no evidence of inflammation



Fig 23—Ichthyosis

Syphilis The scales are dry, and are of a grayish color; they are usually associated with papules and are especially marked on the palms and soles The history and other evidences of syphilis will assist in the diagnosis

Lupus Erythematosus There are two types, the discoid and the disseminated. The lesions are reddish and covered by grayish or brownish scales Upon the face they have a butterfly distribution. There is no itching (SEE: p. 153).

Pityriasis Rosea The scales are found especially on the trunk, and are associated with small rose red macules. There is no itching The disease runs an acute course of a few weeks' duration

Tinea Tonsurans (ringworm): The scales are dry and are few in number, associated with circumscribed red patches which tend to disappear in the center. There is often marked itching Microscopic examination reveals the *Trichophyton*. The tinea tonsurans may invade the skin of various parts of the body; the lesions produced vary somewhat with the affected location

3. **Ulcers:** Many diseases are characterized by the formation of ulcers, either single or multiple, small or large, which may effect any part of the body.

Tuberculous ulcers: These may occur primarily in the skin or they may break through the skin because of tuberculous bone affection or tuberculous glands.



Fig. 24—Lupus erythematosus disseminatus

Diabetic ulcers: These occur generally upon the toes or feet and may be a forerunner of gangrene of these parts

Chancroids: Chancroids usually cause ulceration of the genitals.

Syphilis: The crusts are thick; of a dark brown or green color and are often associated with ulcers which discharge freely. The history and other evidences of syphilis will aid in the diagnosis.

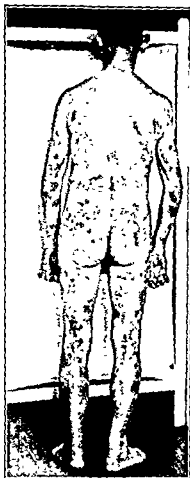


Fig 21—Chronic squamous eczema.

Impetigo: The crusts are thick and yellow, appear stuck on, and are associated with blebs which appear in crops.

Favus: The crusts generally appear on the scalp; they are yellow, brittle, and cup shaped. They are usually perforated by a hair, and have a characteristic musty odor.

Tinea Tonsurans (ringworm of the scalp): In neglected cases the lesions may be associated with crusting. It is usually observed in children. The grayish scales, the dry, brittle, and broken hairs projecting through the crust, the alopecia, and the detection of *Trichophyton,* the causal agent, are the diagnostic features.

2. **Scales:** Scales are dry exfoliations from the upper layers of the skin They are observed in the following diseases:

Squamous Eczema. The scales are usually associated with papules; the underlying skin is red and thickened and there is often marked itching.

Seborrhea Sicca (dandruff): The scales are fine, flaky and greasy and the underlying skin shows *no* evidence of inflammation. The sebaceous follicles are often dilated.

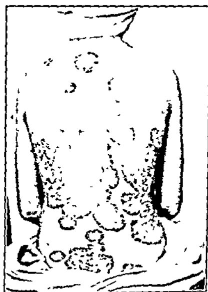


Fig 22—Psoriasis

Psoriasis: The scales are dry, and are of a pearly-white color; they are associated with circumscribed, sharply-defined, elevated inflammatory patches; the extensor surfaces, the elbows and knees, are especially involved. There is little or no itching.

Tularemia: This is caused by infection with bacterium tularense, transmitted by infected rabbits or other rodents. In the ulcerative type punched-out ulcers form at the site of inoculation, i.e., the face, fingers or hands The regional lymph glands become swollen and inflamed and may suppurate It



Fig. 27—Epithelioma

is accompanied by fever which may last for weeks or months. A positive agglutination test in dilutions of 1 to 20 up to 1 to 620 is diagnostic.

Yaws: In the tertiary stage painless granulomatous ulcers covered by a yellowish crust may occur on the extremities The bony structures may become involved The skin and bone lesions of yaws often resemble those of tertiary syphilis (*Gangosa*).

Tropical Ulcers (Tropical Phagedena): These occur most often upon the lower extremities. The ulcers are flat, rounded and may be covered by thick, dirty crusts or by white pseudomembranes They are common among the barefooted nations of tropical climates and occur during the damp season of the year.

Oriental Sore (Delhi Sore): This is caused by the *Leishmania tropica* and is fairly common in Syria. It occurs first as a papule which may later ulcerate and cause a scar (See: p. 1070).

Leishmaniasis Americana (Forest Yaws). The lesions which at first are papular soon ulcerate. They occur on the exposed parts of the body and at times on the mucous membranes of the nose and pharynx. The ulcers have slightly raised and indurated borders and are slightly tender. The surrounding tissue is somewhat inflamed. The regional lymph glands may be somewhat enlarged and tender, occasionally they may suppurate (See: p. 1070).



Fig. 28—Yaws
(Philadelphia General Hospital)

Fungous Infections: Ulcerations also occur in various *fungous infections* such as *actinomycosis, mycetoma* or *madura foot* (See: p. 1093).

Simple Ulcers: These may result from trauma, the application of caustics, or the

Granulosa Inguinalis: This condition causes large ulcerations in the inguinal regions.

Anthrax (malignant pustule): This starts as an inflammatory papule which soon becomes edematous, ruptures and forms a deep discharging ulcer. The regional lymph glands become swollen It is accompanied by high fever and severe systemic manifestations.



Fig 25—Secondary syphilis

Glanders (farcy, equinia, malleus) This is an infectious disease caused by the bacillus mallei. The skin lesion starts as an inflammatory papule or vesicle at the site of infection; it rapidly becomes nodular, pustular and ulcerates. Numerous cutaneous areas may undergo sloughing and ulceration and cause a purulent discharge.

Syphilis: The ulcers are deep and have a punched-out appearance; they secrete an abundant offensive material. They often involve the bone and extend rapidly. They are not painful and the imperfect cicatrix which they produce is soft. The history and other evidences of syphilis will aid in the diagnosis.

Epithelioma: This appears late in life, seldom before 45. There is usually a single center of ulceration, the ulcer being irregular in shape with thickened and infiltrated edges. The secretion is scanty and bloody. The progress is somewhat slow, and in advanced cases there is often pain, and involvement of neighboring lymph glands.

Lupus Vulgaris. This generally appears in early life; there are often several centers of ulceration The ulcers are usually superficial; the edges are not thickened and the progress is extremely slow. The bones are never involved and there is very little secretion. Soft papules often develop in the cicatrix, which is firm and contracted.



Fig 26—Gumma of forehead (Philadelphia General Hospital)

Technic: Firm pressure is made over a portion of the edematous part with the index finger; when the finger is removed, the impression still remains.

Edema is caused by a disturbance of the balance between the amount of fluid exuding from the capillaries and the amount taken up by the lymphatics.

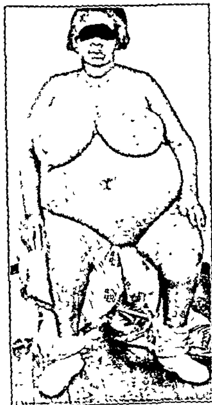


Fig. 29—Anasarca

Varieties: Edema may be *general* or *local*. *General edema* or *anasarca* is caused by venous stasis, altered conditions of the blood as in anemia or hydremia, inflammation; stasis or obstruction, circulatory and cardiac and renal decompensation. It may also be due to starvation, particularly to sodium chloride and protein deficiency (hypoproteinemia).

Local Edema: *This is usually most marked over those portions of the body where the skin is loosely attached. It* usually results from obstruction of the return circulation of a part, thereby causing venous stasis with the resulting transudate. The commonest causes are heart failure and nephrosis. If edema is of cardiac origin, the first evidence of it will be noted in the ankles; usually the patient will state that on arising in the morning the ankles are not swollen, but in the evening or even late in the afternoon, the ankles and often the legs become edematous. The amount of edema is usually directly proportionate to the weakness of the right ventricle. Edema due to renal diseases is first manifested as swelling of the lower eyelids, most noted in the morning on arising, and often disappearing towards the end of the day. As the kidneys become more incompetent, the edema will be generalized. Edema due to hepatic origin is usually first perceptible in the abdomen, and that due to anemia is noted on the dependent parts of the body. Advanced cases of edema, no matter from what etiologic factor, present the same physical signs; namely, swelling and pitting on pressure. Edema due to lymphatic obstruction is usually firmer and does not pit on pressure as readily as that caused by venous obstruction.

Edema Due to Lymphatic Obstruction: Elephantiasis, Hodgkin's disease, myxedema, and edema of nervous and anaphylactic origin, *i. e.*, angioneurotic edema are due to lymphatic obstruction and do not pit on pressure.

Emphysema of the Skin: This condition is caused by the entrance of gas or air into the cellular tissue. The skin usually appears pale, is distended and

action of intense heat or cold. Ulcers are frequently observed on the legs of the aged in association with local nutritional defects and varicose veins. Simple ulcers may be recognized by the history, their location, the appearance of the lesions, and the absence of other symptoms.

Perforating Ulcer of the Foot· This term is applied to a deep-seated ulcer appearing on the sole of the foot; it is most frequently observed in locomotor ataxia. It usually begins as a corn in the neighborhood of the great toe, and is generally associated with anesthesia of the sole of the foot. Ulcers may also occur in the ankles above the external maleolus.

Decubitus Ulcers: This term is applied to bedsores which occur in patients who are obliged to remain in one position for a prolonged period, particularly so in patients who are asthenic or are suffering from grave cerebral or spinal lesions. Bedsores are generally observed on parts which are subject to pressure, as the sacrum, buttocks, calves and heels, and are preceded by erythema and vesication.

III. Scars

Scars on the skin are usually the result of trauma, either recent or old. Scars upon the head and face may be there as the result of a surgical operation or of an accidental injury. Scars on the lips may appear as the result of a chancre, an injury, or following surgical intervention Scars on the face other than those caused by a surgical operation or trauma, may be the result of acne, smallpox, lupus, syphilis, or ulcers. Scars upon the arms and legs may be a result of trauma, or a surgical operation; pinpoint scars over the arms, legs and thighs may follow the use of a hypodermic needle, an important evidence of drug addiction.

IV. Temperature*

The temperature of the skin is usually in keeping with the internal temperature of the body, or with the temperature of an object kept close to it, thus, a hot-water bag applied to the skin will cause a *local increase in temperature* over the part in contact with it, while an ice bag will reduce the temperature of the part with which it comes in contact.

General coldness of the skin is usually caused by poor capillary circulation, as a result of chills and often immediately before death. It may also occur in some febrile diseases when there is weakness or failure of the heart.

Local coldness of the surface may be caused by vasomotor spasms, obstruction of the circulation in localized areas, by venous or arterial thrombosis, and also by exposure to cold

General abnormal heat of the surface is in evidence in almost all febrile diseases. There are, however, some febrile diseases in which the surface of the body is cold and clammy.

V. Edema[1]

By edema is meant an accumulation of serum in the cellular tissue.

Edema of the skin is recognized by *inspection* and *palpation.* On inspection, the edematous part is swollen, the skin covering it, having lost its natural color, appears pale, tense and shining. *Palpation* will elicit loss of elasticity of the affected part, and reveal pitting on pressure

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* For fuller discussion, SEE · *Fever*, pp. 47 to 59

[1] SEE, p 88 and Index.

Technic: Firm pressure is made over a portion of the edematous part with the index finger; when the finger is removed, the impression still remains.

Edema is caused by a disturbance of the balance between the amount of fluid exuding from the capillaries and the amount taken up by the lymphatics.

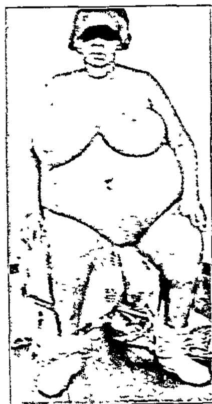


Fig. 25—Anasarca.

Varieties: Edema may be *general* or *local*. *General edema* or *anasarca* is caused by venous stasis; altered conditions of the blood as in anemia or hydremia, inflammation; stasis or obstruction; circulatory and cardiac and renal decompensation. It may also be due to starvation, particularly to sodium chloride and protein deficiency (hypoproteinemia).

Local Edema: This is usually most marked over those portions of the body where the skin is loosely attached. It usually results from obstruction of the return circulation of a part, thereby causing venous stasis with the resulting transudate. The commonest causes are heart failure and nephrosis. If edema is of cardiac origin, the first evidence of it will be noted in the ankles; usually the patient will state that on arising in the morning the ankles are not swollen, but in the evening or even late in the afternoon, the ankles and often the legs become edematous. The amount of edema is usually directly proportionate to the weakness of the right ventricle. Edema due to renal diseases is first manifested as swelling of the lower eyelids, most noted in the morning on arising, and often disappearing towards the end of the day. As the kidneys become more incompetent, the edema will be generalized. Edema due to hepatic origin is usually first perceptible in the abdomen; and that due to anemia is noted on the dependent parts of the body. Advanced cases of edema, no matter from what etiologic factor, present the same physical signs; namely, swelling and pitting on pressure. Edema due to lymphatic obstruction is usually firmer and does not pit on pressure as readily as that caused by venous obstruction.

Edema Due to Lymphatic Obstruction: Elephantiasis, Hodgkin's disease, myxedema, and edema of nervous and anaphylactic origin, *i.e.*, angioneurotic edema are due to lymphatic obstruction and do not pit on pressure.

Emphysema of the Skin: This condition is caused by the entrance of gas or air into the cellular tissue. The skin usually appears pale, is distended and

yields to pressure, though it does not pit. *Palpation* will elicit a creptitation or crackling sound, and *percussion* over that part will yield a somewhat tympanitic note. Subcutaneous emphysema may be caused by the invasion of air-producing microörganisms, or it may occur as a result of rupture of the lung, larynx or trachea. It may also be caused by rupture of the esophagus, stomach and intestines, or by a stab wound penetrating the lungs. Subcutaneous emphysema has often been caused by faulty technic when inducing artificial pneumothorax or pneumoperitoneum.

VI. Moisture of the Skin[1]

The skin under normal conditions has a certain degree of moisture which is not readily recognized by the unaided eye This lends it a definite lustre and softness.

A skin that is abnormally dry, soon becomes hard, brittle and scaling, as is noted in ichthyosis.

Hyperhidrosis or Hyperidrosis (excessive sweating): Pathologically, perspiration is increased in: Rheumatic fever; malarial fever; relapsing fever, septic fevers, pneumonia (at crisis); pulmonary tuberculosis ("night sweats"); Graves' disease; migraine; neuralgia (unilateral sweating); also by certain drugs (opium, pilocarpine, alcohol), and by hot drinks. Local sweating of hands and feet is seen in hysteria, neurasthenia, vagotonia, fright or other emotions, in nervous irritability and in exophthalmic goiter (SEE: p 779).

Anidrosis or Anhidrosis: A deficiency of sweat may be found in cases where an excess of fluid has been withdrawn from the body, as in profuse diarrhea, polyuria, continuous vomiting, severe hemorrhage, diabetes insipidus, myxedema, general anasarca, continued high temperature and in ichthyosis (SEE: p. 59 and Fig. 23, p. 147)

Perspiration may also be altered in color and odor.

Bromidrosis: This is characterized by fetid sweat.

Chromidrosis: Colored sweat, blue, brown, yellow or at times red, is seen in hysteria and in those working in aniline dyes. Yellow sweat is usually due to bile pigment, and is seen in jaundice

Uridrosis: This is perspiration which has a urinous odor; evaporation will reveal white scales or crystals (uremic frost) of urinary solids. This is often found in uremia.

The Mucous Membranes

The mucous membranes, particularly of the mouth, nose and eyes, because of their easy accessibility, are readily studied.

Color

Pallor: This is seen in all forms of anemia.

Temporary Blanching: This occurs in shock, vasomotor spasm, and during severe hemorrhages.

Alternate Blanching and Flushing: This often accompanies aortic regurgitation and aneurysm.

Cyanosis: This is usually caused by asphyxiation, gas poisoning, strangulation and poor circulation, due as a rule to venous stasis or deficient oxygenation

Hyperemia (excessive redness).

1. ***Of the Eyes may be caused by:*** (*a*) Local irritation of the conjunctiva; (*b*) foreign body in the eye; (*c*) ulcer,

[1] SEE: p. 50.

(*d*) any other inflammatory condition of the eyeball and its structure, and (*e*) polycythemia.

2. ***Of the Buccal Mucous Membrane by:*** (*a*) Decayed teeth; (*b*) stomatitis; (*c*) traumatism of any kind; (*d*) scurvy, acute leukemia, etc. (SEE: p 190).

3 ***Of the Nasal Mucosa by:*** (*a*) Ulceration of the nose; (*b*) rhinitis; (*c*) any inflammatory condition of the nasal mucosa.

Jaundice: This is seen in conditions that likewise affect the skin. Often, however, in syphilis, lobar pneumonia, and other febrile diseases, jaundice of the conjunctivae will be noted while the skin remains clear; *per contra,* certain toxic conditions may cause jaundice of the skin while the conjunctivae escape.

Moisture

Excessive Moisture of the Conjunctiva: This occurs as a result of local irritation, or occlusion of the lachrymal ducts.

Excessive Moisture of the Mouth: This occurs in stomatitis; following the ingestion of irritating foods or drugs like pilocarpine; in irritation of the pneumogastric nerve; in certain nervous diseases, in children during teething; and, reflexly, on seeing appetizing food, on smelling pleasant odors, or during sexual intercourse.

Excessive Moisture of the Nasal Mucous Membranes: This is seen in coryza, nasal irritation, ozena, nasal diphtheria, vasomotor ataxia and nasal obstruction, hay fever and other allergic states

Dryness of the Mucous Membrane: This is seen in fevers, severe diarrhea, chronic gastritis and some diseases of the liver. It is often also noted during excitement, shock and severe prostration or in excessive thirst and fatigue.

Rashes

Mouth Rashes: These are caused by stomatitis in any form, *i. e.,* acute catarrhal aphthosis, ulcerative, parasitic, mycotic (thrush), gangrenous; and by secondary and tertiary syphilis; mercurial and corrosive poisons; by foot and mouth disease; diphtheria; Vincent's angina; herpes zoster; pellagra; influenza acute leukemia, smallpox; chickenpox; tuberculosis; measles; scarlet fever, and drugs

Herpes: These are seen on the lips in typhoid fever, meningitis, pneumonia; Koplik's spots are seen in the prodromal stage of measles Mucous patches appear on the lips and in the mouth in secondary syphilis; other lesions that may affect the lips are tuberculous ulcers, cheilitis, chancre, cancer and epithelioma, and accidental injuries.

Petechiae: Petechiae upon the mucous membranes of the mouth are found in scurvy, purpura hemorrhagica, acute leukemia, hemophilia, pernicious anemia, splenic anemia, bacterial endocarditis, trauma, and hereditary telangiectasis.

Pigmented Spots: Pigmented spots in the mouth are found in Addison's disease, argyria and other heavy metal poisonings.

Apigmented* or *White Areas: In the mouth these may be caused by leukoplakia, lichen planus, electrogalvanic lesions caused by artificial dental plates, mucous patches and corrosive poisons.

Lupus Erythematosis Disseminata: This is a constitutional disease of unknown origin, in which lesions resembling the discoid type of lupus erythematosis may appear upon the face and body.

It is commoner among young females than males, and is uncommon in the negro.

Symptoms, Physical Signs and Laboratory Data: (*a*) Fever: The temperature is irregular, long continued and is marked by remissions. (*b*) Arthralgia: Pain in various joints which at times is associated with swelling and fluctuation (polyarthritis) is common. (*c*) Polyserocitis: Pleural, pericardial and at times peritoneal effusions occur in advanced cases. (*d*) Rash: The skin lesions usually are most prominent upon the exposed portions of the body, *i e*, the face (bridge of the nose, cheeks, chin, upper lip and forehead), the exposed portion of the chest, the hands, particularly the ends of the fingers and the thenar and hypothenar eminences. It may also occur upon other parts of the body At *times the rash may* be absent or nondiscernible The lesions consist of erythematous, slightly raised patches of varying size and shape, covered with brownish or grayish fine scales; occasionally there are telangiectatic areas intermingled with these lesions. Upon the face the *lesions assume a butterfly shape* The mucous membrane of the mouth may also become invaded by reddish macules which later form small ulcers. (*e*) Leukopenia: The white cell count may range from 3000 to 6000; there is also a secondary anemia and a low platelet count. (*f*) Hematuria. Red blood cells are nearly always present in the urine; albuminuria is moderate. The complications vary; there may be purpura, various vascular changes as well as peripheral nerve changes.

Erythema Induratum (Bazin's Disease): This occurs as a red or violet, gradually turning brown, discoloration of the skin, in which develop small nodules that may ulcerate and leave depressed lesions covered with a serous exudate. These lesions are bilateral and develop chiefly upon calves of legs, though face, trunk and arms may be involved. It is caused by tubercle bacilli

Erythema Arthriticum Epidemicum (*Haverhill Fever*): *This is a febrile* arthralgic disease characterized by an abrupt onset with chills, fever, malaise, vomiting, headache, polyarthritis and the appearance, chiefly upon the ankles and wrists, of a rubelliform or morbilliform rash which tends to become hemorrhagic. *The temperature curve is marked by a* sudden rise which may last from two to five days followed by a remission in which there is comparative freedom from symptoms, after a few days fever and other symptoms recur. This disease is caused by the *Haverhilla multiformis* which may be recovered from the blood and affected joints of the patient The disease usually occurs *in epidemics. Those in Chester,* Pa, and Haverhill, Mass, were traced to infected raw milk Sporadic cases though rare were traced to rat bites.

Boek's Sarcoid (Cutaneous): This is characterized by the formation, upon the face and upper part of the body, of symmetrically arranged lesions which are deep reddish brown firm nodules varying in size from a pinhead to a walnut. The small nodules occur in groups in the patches of hardened skin especially about the lower lids and chin, they do not suppurate.

Darier-Roussy Sarcoid: This differs from Boek's sarcoid in that the lesions are located beneath the skin, the skin is thicker and the nodules are larger and have a predilection for the trunk and buttocks. However, the lesions may occur *about the ears, nose and cheeks. They* are of a purplish red color. Both varieties occur in the middle-aged.

CHAPTER VIII

Examination and Diseases of the Head, Face, Eyes, Ears, Nose, Mouth and Neck

The Head

In order to be able to diagnose satisfactorily a pathological skull condition, thorough familiarity with the topography of the normal skull is necessary. It should be borne in mind that in health the volume of the brain and the size of the skull bear a constant relation to each other, and as it is possible for a variation to exist in the size of the brain of normal individuals, it naturally follows that normal skulls may also vary within certain limits The male skull is normally larger than that of the female, and in both men and women of certain races there are noticeable differences in size Indeed, the dimensions of the skull form one of the most marked characteristics by which one race may be differentiated from another. What is usually termed "family likeness" is due mainly to the shape of the skull.

The examiner should *not* lose sight of the importance of race characteristics. Occasionally, the variations in the contour of the head due to peculiarities of race may be so pronounced as to cause one to judge them artificial deformities or pathologic changes It is well known also, that the resistance of the Ethiopian skull is so much greater than that of the Caucasian, that a force sufficient to crush the bones of a white man will do no more than traumatize the superficial tissues of a negro

Just as the normal development of the skull is dependent upon a number of factors, so may pathologic conditions of the skull arise from a variety of causes Abnormalities of the skull content—that is, the brain and its coverings—may influence skull contour, and on the other hand, disturbances of the bony covering may lead to abnormalities of the brain

Examination of the Head

The head is examined by inspection, palpation, percussion and auscultation, occasionally also by mensuration, x-ray, encephalography, ventriculography and spinal puncture.

The head is examined for size, shape, signs of injury, mobility, rashes, condition of the hair and the general appearance of the face

Inspection: The skull is inspected for size and shape as well as for the contour of the face. By inspection one may also note the existence of any pulsating areas, and changes in the color of the soft tissue covering the skull. The location of a fracture may be suspected by the presence of a suffusion A greenish tumor, if not caused by an injury, may indicate a chloroma Blue markings caused by distended veins are evidence of a general disturbance of the circulation a condition not infrequently observed in tumors of the scalp or of the vault, and in increased intracranial pressure.

Palpation: This may reveal changes in the structure of the bone, the sense of touch detecting inequalities in the outer surface However, palpation is not always of great value in the diagnosis of

skull lesions or changes, though often it is of service in determining whether an indentation is due to a definite skull defect or to a recently acquired lesion. Abnormal compressibility of the skull may be found in cases where the skull changes are due to insufficient ossification, as seen in old people, in hyperparathyroidism, in Hans-Schuller-Christian's disease, in multiple myelomata, in syphilitics, after comminuted fractures, in scale formation over hematoma and in halisteresis of rickets. Tenderness may be elicited in brain tumors or brain abscesses, inflammation of the soft parts, and neuralgia

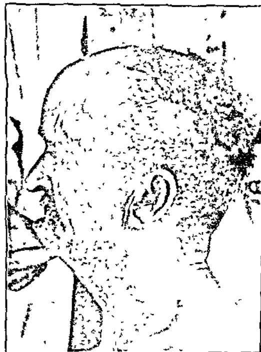


Fig 1—Macrocephalus. (Taking size 8¼ hat.)

Percussion: Percussion does not furnish definite information as to the condition of the skull contents, although testing bone conductivity is often a satisfactory method of discovering pathologic changes in the skull Tenderness elicited by percussion over the sinus regions indicates acute inflammation.

Auscultation: This is of little value in the examination of the head. Pulsation sounds are evidence of the presence of intracranial aneurysms, or narrowing of the lumen of a large intracranial blood vessel.

It is evident that physical examination of the skull is not always a fruitful measure. Radiographic diagnosis is often of greater value. X-ray examination of the skull will reveal the size of the bones and cavities of the skull, the presence of blood vessel forkings and the presence of certain types of tumors. The diagnosis of certain brain abnormalities may be aided by encephalography, ventriculography and by spinal puncture.

Size and Shape of Head

The size and shape of the head and face may be influenced by bone deformity, soft tissue changes, or both. At birth the normal circumference of the head is about 14 inches (35 cm.) and at one year it is about 18 inches (45 cm.).

I. Macrocephalus (marked enlargement of the cranium): This is found in (*a*) hydrocephalus, (*b*) acromegaly, (*c*) rickets, (*d*) osteitis deformans (Paget's disease), (*e*) leontiasis ossea, (*f*) myxedema, (*g*) sporadic cretinism, (*h*) idiocy, (*i*) facial hemiatrophy, (*j*) leprosy, (*k*) congenital syphilis and (*l*) achondroplasia.

(*a*) ***Hydrocephalus:*** The head is usually globular and sometimes pyramidal in shape, the face being disproportionately small. The eyes are directed upward and hidden within prominent sockets; the sutures are widely separated, the fontanels bulging and fluctuating, while the cranial bones are very thin.

(*b*) ***Acromegaly:*** The head is somewhat enlarged but the greatest increase in size is noted in the facial features The malar bones and mandible become prominent, the orbital ridges protrude, while the nose and other soft parts of



Fig 2—Hydrocephalus

the face greatly increase in size; the teeth become widely separated (SEE: Figs. 3 and 4, pp. 764 and 765).

(*c*) ***Rickets:*** In the *rachitic head* the forehead is prominent, the head as a *whole is elongated, square and is flattened* abnormally at the vertex; the fontanels remain open long after the usual time for closure, sometimes up to the third or fourth year of life. The presence of craniotabes is a significant finding in rickets (SEE: pp. 727 and 908).

(*d*) ***Osteitis Deformans*** (*Paget's* disease) : The face is triangular in shape with the base of the triangle upward; the head is lowered and is carried forward, so that the chin rests below the episternal notch; this is usually associated with deformity of other bones of the body (SEE: Fig. 7, p. 728).

(*e*) ***Leontiasis Ossea (hyperostosis cranii):*** This shows enlarged and globular cranium, with prominent malar bones and massive orbital rims

(*f*) ***Myxedema:*** This produces a round "full moon" face, with coarse features, thick nostrils, large mouth and thick lips, causing the head to appear enlarged

(*g*) ***Sporadic Cretinism:*** This is characterized by a large, flat-topped head, with a broad, flat face, a low forehead, widely separated eyes, a flat nose; and the tongue protruding from the mouth which is usually kept partly open



Fig 3—Cretinism (sporadic).

(*h*) ***Idiocy:*** This may often be recognized, not so much by the enlargement of the face as by the peculiar expression found around the eyes, together with open mouth and protruding tongue. The head is usually enlarged, either because of associated rickets or hydrocephalus,



Fig 4—Hydrocephalus with hypopituitarism (Courtesy of Dr. J C. Yaskin.)



Fig. 5—Leprosy.

although it may be very small, as seen in microcephalic idiots.

(*i*) ***Facial Hemiatrophy:*** The face appears as though divided by a longitudinal line; each half having the appearance of belonging to a different countenance; one side of the face is usually smaller than the other



Fig 6—Head and face of achondroplastic dwarf

(*j*) ***Leprosy:*** The ulcerations and cicatrizations resulting from the tuberous growths of leprosy may slowly change the shape and contour of the face so that in time it will assume a leonine aspect.

(*k*) ***Congenital Syphilis:*** The sutures are depressed and surrounded with

protuberances, most noticeable in the frontal region, often giving it a centrally constricted appearance.

(*l*) ***Achondroplasia:*** The head seems large in proportion to the body. The

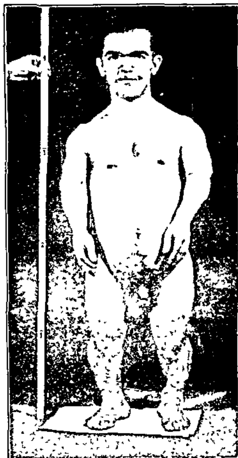


Fig. 7—Achondroplastic dwarf, age 24 years Note Normal size head and trunk Depressed root of the nose, massive muscles, very short upper and lower extremities and normal size genitalia

vault is large, the bridge of the nose is depressed and the chin is prognathous

II. Microcephalus (abnormally small head): This is generally characteristic of idiocy and usually associated with a small brain content. The condition is congenital; the sutures close early. A small head in proportion to the body is also seen in congenital eunuchoidism.

III. Asymmetry of the head: This may occur as a result of systemic disease or because of the presence of local tumors. Acromegaly, rickets, facial hemiatrophy and leprosy may be cited as examples of asymmetry due to systemic disease; local asymmetry of the head is most commonly due to tumors such as sebaceous cyst, sarcoma of the periosteum, syphilitic nodules, ivory exostosis, secondary malignant disease, scleroma (rare), and hematoma.



Fig 8—Microcephalic idiot. (Philadelphia General Hospital.)

Scars and Signs of Injury

Scars upon the head are the result of healed wounds following injury or surgical intervention; or may be caused by

certain skin diseases and syphilitic periostitis.

Nodes may be formed upon the skull as a sequel to some injury during early childhood, or as a result of syphilitic periostitis.

Rashes

Many of the rashes that affect the skin in general, also invade the hairy scalp, several rashes, however, have a predilection for the scalp, *i. e.*, seborrhea sicca, favus, tinea tonsurans, various forms of eczema, chickenpox, some of the syphiloderma, etc. (for a fuller discussion on Rashes (SEE: p. 131).

Posture of the Head

Abnormal Fixity of the Head: In certain pathologic conditions the head may be fixed in an abnormal posture. It is *retracted* in acute meningitis, either suppurative or tuberculous; in meningismus; in cerebral abscess or tumor; in thrombosis of the superior longitudinal sinus; in acute encephalitis; in laryngeal obstruction, especially in children suffering from laryngeal diphtheria; in tetanus; hydrophobia and epilepsy; in spasmodic torticollis; *in strychnia poisoning; in* paramyoclonus multiplex, and in hysteria. Rachitic children show a tendency to keep their heads somewhat retracted, and it has also been noted that normal infants of nervous temperament may assume this position during a violent fit of crying or because of pain.

The head may be flexed in painful lesions at the back of the neck; in lack of muscle support, especially in children, and in fracture of the atlas.

Inability to move the head may be due to caries of the cervical vertebrae, resulting from tuberculosis, traumatism, or any other cause. Disease of the articulation between the atlas and the occiput causes painful deglutition and immobility of the head.

Abnormal fixity of the head, whatever the position, may be due to a postpharyngeal abscess or occipitocervical myelalgia, to arthritis deformans, swollen and painful cervical glands, sprains of the cervical muscles, general traumatism of the neck, or rheumatism. It may also be due to caries of a molar tooth and consequent painful focus of infection, to congenital spasmodic torticollis, to the contraction caused by the cicatrices of burns, or faulty union of muscles or tendons in the neck.

Abnormal Movements of the Head: These may occur as regular noddings or spasms, or they may be present only at irregular intervals, being manifested by a variety of motions *Habit spasms* consist of nodding or twitching of the head, most marked when the patient's attention is called to the abnormality, and disappearing when he is not self-conscious or is asleep. Such head nodding is common in epileptic children. *Rhythmical head nodding* is seen in aortic regurgitation, paralysis agitans and senility.

Spasmodic torticollis consists of spasmodic jerkings of the head occurring every few minutes. The head is usually brought toward one shoulder, the face being turned in the opposite direction and the chin raised, while the shoulder is simultaneously jerked upward to meet the head.

Tonic torticollis is permanent; it is often due to Pott's disease or it may be congenital.

Chorea produces movements of the head which are always irregular and may be of a jerking character or display a variety of motions. The muscles of both the face and arms are likely to be similarly affected.

The Hair

The color, texture and amount of hair varies greatly in different individuals Abundant hair, of good quality and texture, is usually found in robust persons, while dry, coarse, brittle hair is likely to be an indication of general asthenia, or of some local pathological condition of the scalp.

About the fortieth year the hair usually begins to turn gray, especially about the temples, and becomes progressively grayer as age advances Premature graying of the hair may be hereditary; in certain families some of the members become gray at 20 or even younger. Early graying of the hair is often also associated with premature senility and other degenerative changes. Whitening of the hair has been observed in those who have been subjected to a sudden fright and prolonged terror; anxiety and intense nervousness have been known to produce premature grayness Discoloration of the hair may at times be caused by the handling of certain dyes, and has been seen in those who work with copper, cobalt and indigo.

Hypertrichosis: Abnormal growth of hair may be either congenital or acquired. The cause is often obscure. It is found in association with certain endocrinopathies, as in hyper- and, at times, in hypopituitarism, in hypergonadism and in hyperadrenalism A luxurious growth of hair upon the head has frequently been seen in persons who have been confined to their beds for a year or two suffering from pulmonary tuberculosis, although previous to the onset of the disease their hair had been of an indifferent quality.

Atrophy of the Hair: This may be due to local scalp conditions; to systemic affections, such as cachexia, myxedema, extreme emaciation, or sometimes, tuberculosis; and may follow a prolonged illness. This condition has also been observed as a sequel to focal infections, *i. e*, in tonsils, teeth or some other part of the body.

Alopecia (baldness): This may be general or circumscribed. *General baldness* in middle life frequently has its origin in eczema, seborrhea or favus of the scalp during childhood Often there is no discoverable cause for the condition. *Congenital alopecia* may have an endocrine basis, possibly of pituitary origin. Acute fevers, toxemia, syphilis, myxedema, also certain cutaneous diseases of the scalp, and anemia may produce either general or local baldness *Local baldness* (alopecia circumscripta or alopecia areata) has been noted as a result of tinea tonsurans, syphilis, scars, or other local scalp infection The baldness on the back of the head so often



Fig. 9—Congenital alopecia (Courtesy of Dr. N H Winkelman)

noted in rachitic children is due no doubt to the constant rolling of the head back and forth upon the pillow. The alopecia after fevers, like typhoid, is usually temporary.

The Face

The face should be studied as to its size, color and condition of the skin, and the general expression, whether of intelligence, pain, surprise, worry, fright or any other visible emotion. Certain diseases leave an indelible impression upon the countenance, and in a certain few the expression is so characteristic as to be almost diagnostic

Expression

Mouth Breathing: **This usually** causes the individual to develop a stupid expression, with the mouth partly open, the nose apparently "stopped up" and the eyes somewhat protruding and unintelligent in expression

Chronic Alcoholism: This presents an absent, vacant facial expression, tremors about the corners of the mouth with enlarged superficial capillaries around the nose and cheeks, giving the typical "red nose" of the alcoholic

Drug Addiction: This usually produces pinched features, "shifty" eyes, and tremors of the lips and facial muscles

Abdominal Diseases: The patient bears an anxious look, the features are pinched, the general expression being *one of anxiety and apprehension.*

Facial Hemiplegia: This causes a drooping of one corner of the mouth and a *smooth, nonwrinkled appearance* of the affected half of the face. The mouth is drawn towards the sound side. The lips cannot be puckered, and *an attempt* to whisper labial sounds causes bulging of the cheek.

Insular Sclerosis: This gives a facial appearance of fatuousness and flaccidity, with a *vacant stare, the patient appearing* to take no interest whatever in his surroundings.

Cretinism: The cretinoid face is broad, the nose is broad and flat. The lips are thick and the ears coarse, while the



Fig. 10—Myxedema (B M. R minus 32) resembling Myasthenia Gravis (Philadelphia General Hospital.)

mouth is *generally held open, the tongue* usually protruding There is a small and undeveloped chin, brittle, scanty hair, and coarse skin, which is dry and of a brownish yellow *tint.*

Myxedema: The general expression of the myxedema face is *one of apathy* and *stupor;* the skin is coarse, thick, dry and sallow; the cheeks are occasionally cyanotic, the eyelids puffy, *while the nose is broad and the* ears are thick. The lips, especially, are exceedingly large and turn up so that they expose a part of the

mucous membrane of the mouth. The hair is scanty and the eyebrows are poorly marked.

Congenital Syphilis: This presents a typical face. The forehead appears overhanging, the nasal bridge is depressed, scars or deep fissures often radiate from



Fig 11—Exophthalmic goiter

the corners of the lips, the complexion is sallow, the eyes are often diseased, and the teeth have the characteristic Hutchinson's notches and narrow edges and are widely interspaced

Exophthalmic Goiter (Graves' disease) The general appearance of the face is that of one having been thoroughly frightened, the eyes stare and protrude somewhat (SEE: p 777)

Myopathic Face: This is due to atrophy of the facial muscles The characteristics of this face are usually found around the mouth and are noted in the loose pout of the lips and the "twisted" character of the smile The deformity of the face usually depends upon the particular group of muscles atrophied, resulting in ptosis of the upper eyelids, or an inability to whistle, or to blow out the cheeks.

Myasthenic Faces: These are of two types: In one, the patient when asked to smile, will have a normal smile on one side of the face and a sneer on the other. in the second type, the upper eyelids are apparently closed, the mouth is partly open, and the patient continually has the appearance of being exhausted with fatigue.

Paralysis Agitans (Parkinson's syndrome): The features are set, and the general expression has the appearance



Fig 12—Parkinson's syndrome, postencephalitis lethargica

of a mask. The eyes, however, appear extremely mobile, often unusually intelligent, seemingly trying to compensate for the immobility of the rest of the face

Encephalitis Lethargica: The patient is somnolent, stuporous and thoroughly relaxed (wax-like flexibility). In

some instances muscular hypertonia or rigidity, coarse tremors and choreo-athetoid movements replace the extreme flexibility. The eyes are closed and the face bears a *tired, annoyed, sleepy expression*. Among the sequelae of this disease is a postencephalitic syndrome which resembles Parkinson's syndrome (paralysis agitans), *i. e.*, a mask-like expression of the face with very alert eyes. There is scanty, mouse-colored hair, and *a florid or mottled complexion*.

Acute Diffuse Peritonitis: This is characterized by an expression of extreme anxiety, the teeth being uncovered by the raised upper lip. The "Hippocratic countenance" (facies of impending death) is well marked.

Dyspnea: This produces an anxious facial expression; the face is cyanotic.

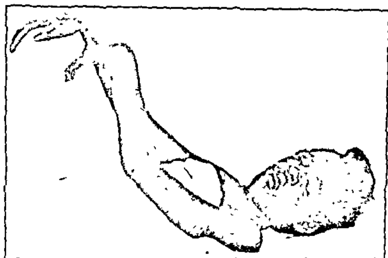


Fig. 13—Lethargic encephalitis with cataleptic phenomena.

Locomotor Ataxia: This causes the *face to assume the following characteristics*: Apparent ptosis of the upper eyelids; wrinkling of the forehead; *inequality of the pupils; sallow complexion, and, at times, drooping of the angles of the* mouth.

Acromegaly: This produces large superorbital ridges, and prominent malar bones; *the nose especially is very large, the lower jaw heavy, the lips thick* and the teeth widely separated.

Mongolian Idiocy: The head is usually *brachycephalic, the nose broad* and flat, the eyelids often inflamed, the ears large and the lips fissured The mouth is kept open with the tongue protruding, as if it were *too large for the mouth* the mouth is open, the lips and tongue *are dry, and the nostrils dilate widely* with each inspiration

Hysteria: This displays its characteristic facies in the expression of extreme pleasure and the amiable smile which are in evidence when humored, but at once changes to a frown of displeasure when antagonized. In hysterical coma the face is immobile but the color remains natural. When an attempt is made to raise the upper eyelid there is great resistance and quivering.

Pulmonary Tuberculosis (late stage): The face is emaciated, and presents a red flush upon the malar bones, the remainder of the face being very pale; the eyes are widely open and

bright, often with an appealing expression, denoting an unusual degree of intelligence. The alae nasi play during respiration.

Lobar Pneumonia: This causes a deep flush to spread over the entire face, which is often noticeably deeper on the



Fig. 14—Parotid tumor

side of the affected lung. The hurried respirations cause continuous playing of the alae nasi.

Renal Disease (acute and chronic parenchymatous): The face is pale, almost ghastly, with general puffiness and marked swelling under the eyes.

Typhoid Fever: During the acme stage the patient presents a *dull and* apathetic appearance, the tongue is dry, the teeth are often covered by sordes, the mouth is kept slightly open, and the lips are dry and fissured (typhoid state).

Hippocratic Facies: A common designation of the face before impending dissolution is marked by the hollow appearance of the eyes, the extreme sharpness of the nose, the collapse of the temples, and the contraction of the ears, so that the lobes turn outward The skin of the face assumes a dark-brown, leaden or livid hue.

Facial Coloring

The color of the face may be the same as that of the body, or it may assume a sallowness, flush or any other discoloration.

Sallowness: This is a peculiar combination of pallor with a brownish yellow tint It may be normal to brunettes or to the natives of hot climates In others the appearance of sallowness should arouse suspicion of some pathologic condition. Sallowness is observed in cachexia, syphilis, malaria, chronic gallbladder disease, lead poisoning, cancer,



Fig. 15—Sarcoma of parotid.

certain anemias (particularly in brunettes), Addison's disease, and in arthritis deformans. It is also likely to be observed in those who are habitually constipated, and in those suffering from gastric disorders due to hepatic, pancreatic or enteric diseases.

Brown or Brownish Yellow Spots (liver spots): These are often noted in pregnancy (chloasma uterinum), in malignant affections of the uterus or liver, and in exophthalmic goiter. Certain irritants like mustard, turpentine, etc., and



Fig. 16—Tumor of parotid (sarcoma).

the use of cosmetics may cause discoloration of the face. Sunburn and exposure to the weather often cause irregular yellowish brown spots (freckles) upon the skin.

Flushing (hyperemia): This may be either evanescent or persistent. *Evanescent flushing* may be due to such emotions as joy, shame or fear. *Persistent flushing* may be caused by various febrile diseases, by pulmonary tuberculosis, as already noted, by convulsions (during the seizure), by alcoholism, by the presence of large abdominal tumors, by a large tumor or a goiter partially interfering with the circulation and by wearing tight collars Plethoric individuals, and those having hypertrophied hearts often present flushed faces. Flushing is also noted in polycythemia vera, Ayerza's disease, chronic pulmonary fibrosis, and in certain types of congenital heart disease.

Alternate redness and pallor of the face is frequently seen in cerebrospinal meningitis, typhoid fever, in certain



Fig. 17—Adenolipomatosis

vasomotor conditions, and during the menopause

Cyanosis: This may be congenital or acquired *Congenital* cyanosis may be

caused by malformations of the heart, *i e*, pulmonary stenosis, patent interventricular septum, patent foramen ovale and congenital constriction of the larynx, trachea or large bronchus. *Acquired* cyanosis may be the result of asthma, whooping cough, pulmonary tuberculosis, advanced emphysema, dilated right heart, croup, obstruction of the trachea from without or from within, aneurysm, tumor, foreign body, goiter, polycythemia, asphyxia and drug poisoning (coal tar, chloroform, etc).

Edema

Edema or swelling of the face is often noted in renal, cardiac and blood diseases which cause general anasarca Certain chest diseases, particularly pneumotherax, mediastinal tumors and aneurysm, will often cause puffiness of the face on account of their interference with the return circulation

Localized Edema: Evanescent edema may be caused by urticaria, anaphylaxis or angioneurotic edema Swelling and puffiness of the forehead may occur in glanders and in thrombosis of the superior longitudinal sinus

Swelling of the Upper Jaw: This may be due to alveolar abscess, parotitis or parotid tumor, necrosis of the bone or disease of the antrum, carcinoma and sarcoma.

Swelling of the Lower Jaw: This is usually caused by alveolar abscess, actinomycosis, occasionally by obstruction of a salivary duct, or the presence of a cyst, sarcoma or gumma.

Swelling in front or behind the ear (when not due to mastoid disease) when it extends downward to the angle of the jaw, either unilateral or bilateral, is due to mumps The cheeks may also become swollen on account of inflammation of the gums, as is seen in scurvy, gangrenous stomatitis and anthrax.

Facial Spasms

Spasms of the facial muscles may be continuous or intermittent, unilateral or



Fig 18—Edema of the face.

bilateral, affecting one, or a number of muscles at the same time. Spasms of the facial muscles may be caused by disease of the teeth, skin, eyes, nose, or by some constitutional or nervous disorders.

When facial spasm is observed the following possible types should be considered:

Brown or Brownish Yellow Spots (liver spots): These are often noted in pregnancy (chloasma uterinum), in malignant affections of the uterus or liver, and in exophthalmic goiter. Certain irritants like mustard, turpentine, etc, and



the use of cosmetics may cause discoloration of the face. Sunburn and exposure to the weather often cause irregular yellowish brown spots (freckles) upon the skin

Flushing (hyperemia): This may be either evanescent or persistent. *Evanescent flushing* may be due to such emotions as joy, shame or fear. *Persistent flushing* may be caused by various febrile diseases, by pulmonary tuberculosis, as already noted, by convulsions (during the seizure), by alcoholism, by the presence of large abdominal tumors, by a large tumor or a goiter partially interfering with the circulation and by wearing tight collars. Plethoric individuals, and those having hypertrophied hearts often present flushed faces. Flushing is also noted in polycythemia vera, Ayerza's disease, chronic pulmonary fibrosis, and in certain types of congenital heart disease.

Alternate redness and pallor of the face is frequently seen in cerebrospinal meningitis, typhoid fever, in certain



Fig 17—Adenolipomatosis.

vasomotor conditions, and during the menopause.

Cyanosis: This may be congenital or acquired *Congenital* cyanosis may be

also be either unilateral or bilateral, and affect either one muscle, or a whole group of facial muscles.

Facial Paralysis

Paralysis of the face is usually unilateral; rarely, bilateral. In order to determine whether or not facial paralysis exists, the following is to be noted:



Fig 20—Bell's palsy.

When the forehead is wrinkled, the affected side of the forehead remains smooth; when the eyes are shut, the one on the affected side will remain partially open; when attempting to whistle, there will be no puckering on the affected side of the mouth

When the patient blows through the mouth, most of the air will come out of the paralyzed side, and in eating, the food frequently escapes through the same side.

Paralysis of the face may be of *peripheral* or *central origin*. If it involves the facial nerves only, not constituting part of a more general hemiplegia, it will present the following characteristics: The eye cannot be completely closed; the forehead cannot be wrinkled; the tongue does not deviate from the middle line (*Bell's palsy*).

If the paralysis is of central origin, the facial nerve is but slightly affected, and the eye on the affected side can readily be closed; the forehead can be wrinkled and the tongue, when protruded, will be found to deviate toward the paralyzed side.

Bilateral facial paralysis is an extremely rare condition; when present, it may be the result of a tumor or gumma at the base of the brain; of disease of the pons or the basilar artery; or it may result from diphtheria, multiple neuritis, double mastoid disease, or bilateral and symmetric cortical lesions

The Eyes

When examining the eyes, the following should be noted· The condition of the eyelids, of the conjunctiva, the sclera and the cornea, the reaction of the pupils and their relation to each other; the state or tension of the eyeballs; and, when possible, an ophthalmoscopic examination of the retina should be made

The Eyelids

The patient should be placed in a good light and the surfaces of the *lids* examined for swollen superficial veins and edema, and the edges for inflammation, parasites, misplaced cilia or foreign bodies.

Puffiness or Swelling: This condition, particularly of the lower lid, is noted in renal diseases, cardiac diseases after failure of compensation, the various anemias, angioneurotic edema, arsenical poisoning, cerebral thrombosis, and ecchymosis due either to external traumatism or to strain (often seen in per-

Mimic Spasm: This condition usually occurs in adults, and is more or less constant. It may be either bilateral or unilateral, and is accompanied by the partial closing of the eye on the affected side.

Habit Spasm: This condition is common in young girls from 7 to 14 years of age. The spasm usually consists of sudden winking of the eye, rapid one-sided contraction of the mouth, sudden drawing down of the upper lip between the teeth, with continuous protrusion of the tongue so as to touch the upper lip, and sniffing, followed by the drawing down of the upper lip on one side. The condition is intensified by emotion.

Convulsive Tic (Gilles de la Tourette's disease). This presents three distinct phases: (*a*) *Coprolalia*, irregular movements of the face or arm, accompanied by associated explosive profane or obscene utterances; (*b*) *echolalia*, muscle twitching accompanied by involuntary repetition of words as they are spoken by others; (*c*) *echokinesis*, constant mimicking of an action performed by another.

Choreic Spasm: Convulsive irregular involuntary jerking movements of the facial and other muscles.

Winking Spasm: Constant and regular clonic contractions of the orbicularis palpebrarum.

Blepharospasm: Persistent closure of the eyes due to spasm of the orbicular muscles may result from disease of the eyes, photophobia, or disease of the orbicularis palpebrarum or from any affection of the nerves supplying those muscles.

Clonic Unilateral Spasm: This type of spasm of one or more facial muscles is caused by pressure upon, or irritation of, the facial nerve.

Miscellaneous Facial Spasms: Facial spasms are also noted in:

Exophthalmic Goiter (Abadie's sign): This often presents constant successive and rapid raising of the upper eyelids.

Epilepsy (petit mal): Tonic spasms are followed by clonic spasms of the facial muscles.



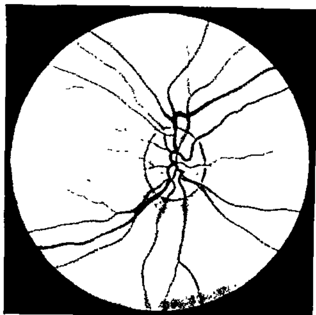
Fig. 19—Amyotrophic lateral sclerosis with bulbar palsy

Meningitis: Spasm of the eyelids, upper lip, chin, or the muscles of either cheek is often observed in the early stages of this disease.

Tetanus: Tonic spasms of the different facial muscles are sometimes observed in this disease (lockjaw).

Spasm Following Paralysis: When the paralyzed muscles begin to recuperate, tonic and, sometimes, clonic spasms may occur in the face.

Tic Douloureux: This often gives rise to spasmodic movements of the face,



NORMAL FUNDUS

The disk is vertically oval with well-defined margins and clearly outlined scleral and pigment rings The color is distinctly brighter in the temporal than in the nasal side. There is a shallow excavation in the center recognized by the light color at the point of emergency of the vessels These are clearly distinguished from each other, the arteries are narrow and brighter, and they have a distinct light streak; the veins are larger, darker and without light streak The pigment epithelium is so laden with pigment that it entirely conceals the choroidal vessels (Uniform or retinal fundus) A circle of darker pigment is concentrated around the disk. The macula is seen with the direct method and appears darker in color than the rest of the fundus with a light reddish area in the center There are no macular or foveal reflexes In other eyes a central, brilliant, foveal reflex can be clearly detected (Adams). (Troncoso's "Internal Diseases of the Eye and Atlas of Ophthalmoscopy " F A Davis Co, Philadelphia, Pa)

tussis, severe vomiting, etc.). Puffiness of the eyelids associated with iritis is noted in syphilis, in glanders and in severe conjunctivitis due to any cause.

Inflammation: ***Hordeolum (stye):*** This is a painful abscess at the edge of the eyelid, usually due to an infection of a hair follicle. Its presence may be an *indication of eyestrain, or of external* infection; it may also occur as a result of some systemic condition

Mucocele (chronic dacryocystitis): This is a chronic catarrhal inflammation of the lacrimal sac, causing it to protrude. This cystic swelling usually appears at the inner canthus of the eye; it is due to stricture of the nasal duct with consequent accumulation and decomposition of tears

Blepharitis: This is an inflammation at the edge of the eyelids, causing them to become red, thickened and encrusted with dried secretions. This condition is found in conjunctivitis, measles, in certain catarrhal affections of the eye, and also as a result of vitamin deficiency and as an allergic phenomenon

Ulceration: *Verrucae* or *warts* on the eyelids, if occurring in elderly subjects, should arouse suspicion of epithelioma. *Ulcers* may be due to two causes which are of especial importance

1. ***Epithelioma*** of the eyelid is an affection of the middle-aged and elderly; it is often of slow development, remaining stationary for years. As a rule, it will be found on the nasal side of the lower lid as a shallow ulcer, covered by a scab, which reveals a raw surface when removed and soon re-forms, without any attempt at healing.

2 ***Syphilis:*** A chancre at times appears upon the eyelid in the shape of a small, moist, slightly ulcerated area, with considerable induration and swelling A positive diagnosis of this condition can only be made by dark-field study and blood tests. *Tertiary syphilitic lesion* of the eyelid is rare; when it does occur, the surface will present an inflamed, indurated and "punched out" appearance

Other Lesions: Xanthomata, which may be flattened or raised are often found near the canthi. Cysts, fibromata and other lesions may affect the upper or the lower lids or the tissues adjacent to the eyes

Movements: ***Blepharospasm:*** This is an involuntary contraction or twitching of the whole or part of the eyelid, which may be due to eyestrain, habit spasm or nervous irritability.

Lagophthalmos: This is a condition in which it is impossible to close the eye completely. This may be caused by the contraction of a scar of the eyelid, and by atony of the orbicularis palpebrarum, facial paralysis, tumor or abscess of the orbit, orbital hemorrhage, fracture of the base of the skull, exophthalmic goiter or by other conditions causing exophthalmus. Incomplete closure of the eye during sleep is often noted in healthy children, and in adults who are greatly exhausted Rolling up of the eyeball and incomplete closing of the lids is frequently seen in hysteria.

Ptosis (drooping of the upper eyelid) This is due to paralysis of the levator palpebrarum and depends upon some interference with the function of the third nerve, either central or peripheral It is also noted in hysteria, in tetanus, in paralyzing lesion of the sympathetic nerve, or in direct traumatism. *Congenital ptosis* which occurs from paralysis, or from defective development of the levator palpebrae superioris, is usually bilateral, while the acquired form is unilateral *If acquired ptosis is due to*

paralysis of the sympathetic nerve, the pupil will be contracted and vasomotor paralysis be manifest on the affected side of the face. Ptosis due to paralysis of the oculomotor nerve usually causes dilatation of the pupil Ptosis, single or double, may occur in tabes dorsalis, facial paralysis, tuberculous meningitis, en-



Fig 21—Horner's syndrome.

cephalitis lethargica, myasthenia gravis, Mikulicz's disease and cerebral tumors, particularly of the corpora quadrigemina and of the pineal body Ptosis may also result from local eye conditions, such as trachoma or disease of the eyeball

Benedict's Syndrome. Ptosis on the side of the lesion associated with a slow rhythmic tremor of the extremities on the opposite side This is found in tumors of the tegmental region of the crus or pons when the red nucleus is involved

Weber's Syndrome Ptosis on the side of the lesion and hemiplegia on the opposite side This is significant of a tumor of the ventral region of the crus cerebri.

Horner's Syndrome: Unilateral ptosis, with contraction of the pupil, recession of the eyeball, and dryness, heat, redness or edema on the same side of the face This is due to paralysis of the cervical sympathetic caused by pressure of a tumor, abscess, enlarged substernal thyroid, subclavian aneurysm, enlarged cervical glands or by direct injury to the cervical sympathetics.

Ptosis Adiposa (false ptosis) and *Blepharochalasis* (relaxation of the eyelid, known also as *dermatolysis palpebrarum*) : These are congenital anomalies, due primarily to defective attachment of the integumenta to the upper margin of the tarsus and the tendon of the levator; the skin cannot be raised with the lid, and hangs down like a pouch over the palpebrae, producing a marked deformity. *Lipomatosis* (lipoma of the eyelid) is allied to these conditions, and is sometimes termed ptosis adiposa.

Ectropion (eversion of the lid margin): This may be caused by relaxation of the skin and tarsus, as is often seen in the aged; or it may take place because of a cicatrix following trauma or infection as in trachoma. Palsy of the facial nerve may also be a cause of eversion of the lower eyelid

Entropion (inward curling of the eyelid): This is often seen in the lower lid because of some spastic contraction of the muscular fibers or of a cicatrix

Adhesions: ***Symblepharon:*** This is an adhesion between the eyelid and the eyeball; it may develop as a result of scars from burns or ulcerations

Ankyloblepharon: This means adhesion between the free edges of the lids.

Epicanthus: This is a crescentric fold of skin which surrounds and par-

Discoloration: A yellowish discoloration of the conjunctiva is seen in obstructive jaundice, hemolysis and certain fevers. It may also be caused by fatty deposits. A bluish white or pearly discoloration is observed in anemia, frequently in nephritis and phthisis. Sky-blue discoloration is often noted in whooping cough and pale conjunctivae in the anemias.

Dryness and Moisture: *Dryness:* In some forms of convulsions, in collapse, and in the typhoid state, the eye may become abnormally dry. Excessive dryness of the eyes is also noted in those conditions which are associated with lagophthalmos. In infants and young children during the course of a severe illness the conjunctiva is dry, when moisture or tears appear it is an indication of beginning recovery.

Abnormal Moisture: This may occur as a result of inadequate drainage such as is produced by blocking of the lacrimal ducts; it is also frequently found in any irritation or inflammation of the conjunctiva, which may be caused by the presence of foreign bodies, or by such diseases as measles, influenza, whooping cough, hay fever, and trifacial neuralgia

The Cornea

The cornea is a transparent coat occupying the anterior fifth of the eyeball In health it presents a pearly-white appearance Pathologically the following conditions may occur·

Arcus Senilis: This is an ill-defined grayish ring circumscribing the cornea, a condition usually found in the aged, or in those suffering from arteriosclerosis or chronic nephritis.

False Arcus Senilis: This is a sharply delineated ring of a clear yellow or yellowish white color, caused by a deposit of fat; as a rule it is of no diagnostic significance.

Keratitis (inflammation of the cornea): In *interstitial keratitis* the cornea assumes the appearance of ground glass, here and there showing small clear areas, through which the pupil may be indistinctly seen. The condition is commonly caused by syphilis or tuberculosis.

Ulcer of the Cornea: This is a break in the continuity of its surface and is often associated with pain, inflammation and photophobia It may be caused by trauma, or by the absorption of certain toxic substances, and is frequently found in exophthalmic goiter, and may also be found in various other constitutional diseases.

The Sclera

The sclera is normally of a bluish white color Deep yellow discoloration occurs in obstructive jaundice; faintly yellow icteroid tinge in cholecystitis without obstruction and in certain febrile conditions

The Iris

The color of the irides may vary from light blue to gray, or they may be brown, yellowish or greenish. In the newborn the irides are of a light blue grayish tint

Chromatic Asymmetry: Difference in the color of the two irides in the same individual occasionally occurs One iris may be blue or gray, while the other may be brown. This condition is consistent with good health, though it is frequently observed in persons who have a neuropathic tendency. Several members of the same family may show this anomaly. Pathologically, chromatic asymmetry may occur in early iritis or cyclitis.

Piebald Irides: Irregularly shaped areas of dark discoloration in one or

tially covers the internal canthus. This condition is normal in the Mongolian race and in many newborn infants of the Caucasian race. Among whites it gradually disappears as the bridge of the nose becomes more fully developed.

Discoloration of the Eyelids: This may be observed in brunettes, particularly at the menstrual period and in early pregnancy. Such duskiness is also observable after fatigue, mental excitement, loss of sleep, severe exhaustion and strain

The Conjunctiva

The conjunctivae are examined by inspection. In order to inspect the conjunctiva thoroughly, both the palpebral and ocular portions should be exposed. In inspecting the lower lid, the examiner's index finger is placed over the lower margin, drawing the lid downward, while the patient is instructed to look up. The conjunctiva of the upper lid is inspected by everting the lid according to a procedure which consists in having the patient turn the eye downward while the examiner gently seizes the central eyelashes of the upper lid between the index finger and thumb of the left hand, the lid is then being drawn downward away from the ball of the eye. The point of the index finger or thumb of the right hand is placed above the tarsal cartilage of the lid which is to be everted, the remaining fingers being steadied on the patient's brow, and by a quick movement the edge of the lid is turned over the point of the thumb or index finger, while this is simultanously depressed.

The upper lid may also be everted by substituting a probe, toothpick or matchstick (if nothing better is at hand) for the thumb or index finger of the right hand. The beginner may find this procedure less difficult, though the technic first described is the more practical.

The conjunctivae are examined for color, degree of moisture, and for the presence of foreign bodies; and for petechial hemorrhages often seen in bacterial endocarditis and in septicemia. *Inflammation of the conjunctiva* is characterized by injection of the conjunctival vessels, lacrimation and photophobia.

Pathologic Conditions: ***Infectious or Catarrhal Ophthalmia*** (pink eye) · The conjunctiva becomes reddened, the vessels are engorged and photophobia is a prominent symptom.

Ophthalmia Neonatorum: This is a gonorrheal conjunctivitis in the newborn; it is infrequently seen in adults and occurs as purulent blennorrhea

Follicular Conjunctivitis: This is a condition in which the conjunctiva of the lower lid is studded with small transparent lymphoid follicles.

Trachoma: The conjunctivae are studded with enlarged follicles, situated on the undersurface of the upper lid, and in the upper conjunctival fornix Thickening and edema of the upper lid, with partial ptosis, are the usual symptoms. The lower lid may also be affected

Pannus: This is a vascular opacity of a part of the cornea In this condition round raised masses, yellowish in tint, appear at the corneosclerotic margin, surrounded by localized areas of vascular conjunctivitis

Membranous Conjunctivitis: This may be due to infection by diphtheria bacilli, or staphylococci The lids are swollen, inflamed and membranous.

physiologically normal finding, or it may be found in the presence of an aneurysm, in disease of the nervous system, head trauma, disseminated sclerosis, focal brain lesion or paretic dementia; sometimes it is seen in locomotor ataxia. The pupils are often unequal in cases of widely dissimilar refraction, and in unilateral blindness. A phenomenon often seen in the early stages of insanity is a varying inequality of the pupils; each pupil independently alternating in dilatation and contraction. In the normal eyes inequality of the pupils will be noted when one eye is exposed to a strong light and the other is in shade

Technic for Testing Pupillary Response to Light: The patient is to face a bright light. The examiner shades the patient's both eyes with his hands or a card and directs the patient to keep his eyes open The shade is suddenly withdrawn so that the light instantly strikes the unshaded eye, and the effect of the light upon the pupil is observed The same procedure is carried out for the other eye An artificial light, such as a pocket flashlight or any other light, may be used as a substitute for sunlight Normally, the pupils contract when exposed to light and dilate when in the dark

Technic for Testing for Accommodation: The patient is asked to fix his gaze upon the examiner's finger, pencil or any other object; the object upon which the patient gazes is gradually removed to some distance in his line of vision, and then it is gradually approached to within a few inches of his eye The reaction of the pupils should be observed when the object is near the eyes and when it is at a distance. Normally, the pupils contract when focused upon near objects and dilate when focused on distant objects

Pupillary Reflexes: ***Mydriasis:*** This is extreme dilatation of the pupil.

Myosis: This is contraction of the pupil The pupil usually contracts when a light is thrown on the retina, and dilates when the light is withdrawn The pupil contracts when any object is brought close to the eye, and dilates as the object is removed to a distance.

Argyll-Robertson Pupil: This does not react to light, but does react to convergence and accommodation. This phenomenon occurs in locomotor ataxia, and is also observed in cerebrospinal syphilis and paresis of the insane.

Accommodation Iridoplegia with Preserved Light Reflex: This is the opposite of Argyll-Robinson pupil. The pupil reacts to light but not to accommodation This condition may occur as a result of a lesion in the oculomotor nucleus, as of postdiphtheritic cyclopegia (paralysis of the ciliary muscle). Unequal contraction or irregularly contracted pupil is often seen in iritis, tabes paresis, posterior synechia and adhesions of the lens.

Immobile Pupil: This is one which does not react to light nor to accommodation

Hemiopic Reflex: In this the pupil contracts when light is thrown on the healthy side of the retina. It does not contract when light is thrown on the paralyzed half

Ciliospinal Reflex: This is a dilatation of the pupil when the neck on the same side is irritated This reflex is absent in glaucoma, general paresis, atrophied iris, and posterior synechia

Westphal Pupil: This is a turning of the eyeball upward and contraction

both eyes should not be mistaken for foreign bodies in an inflamed eye, nor, conversely, should foreign bodies be mistaken for a piebald iris

Iritis (inflammation of the iris): This is recognized by discoloration A blue or gray iris may become greenish, or of a muddy hue, with the pupil contracted and responding sluggishly to light, while a narrow zone of hyperemia encircles the cornea. An iris normally brown does not change color when inflamed Iritis is likely to occur in rheumatism, gout and secondary syphilis

The Pupil

In health the size of the pupil varies with the extent of its exposure to light, and the degree of accommodation and convergence When the eye is exposed to a strong light the pupil contracts, in the dark, the pupil dilates When the eye is first focused on a near object the pupil contracts, when the focus is on a distant object, it dilates The average diameter of the pupil is 4 to 5 mm, normally both pupils are equal.

Mydriasis (dilatation of the pupil) Both pupils may become dilated as a result of the nonconductivity of light Dilatation of the pupils also occurs in fright or other sudden emotion, in anemia, nervous depression and in the first and third stages of anesthesia; and it may be due to the administration of such drugs as belladonna, hyoscyamin, cocaine, etc. It is also observable in coma, hysteria, botulism and irritation of the cervical sympathetic nerve In high myopia (nearsightedness) the pupils are dilated.

One or both pupils may be dilated under the influence of a local mydriatic, and the same phenomenon occurs in the presence of glaucoma, cataract, optic atrophy, orbital disease, brain and spinal cord lesions, and paralysis of the third nerve. Slight unilateral mydriasis is often seen in pulmonary tuberculosis, in aneurysm of the aorta or the innominate artery, or in tumor of the neck causing irritation of the cervical sympathetic nerve. Scratching or tickling the side of the neck often causes one or both pupils to dilate

Myosis (contraction of the pupil) This may be caused by irritation of the oculomotor system, or by paralysis of the dilators Myosis occurs in congestion of the iris, in certain fevers, in the early stages of meningitis, in typhus; because of the local application of a myotic, and in poisoning by such drugs as opium, eserine, pilocarpine, etc Contraction of the pupil may be seen in mitral regurgitation after failure of compensation, in venous obstruction and in pulmonary congestion It is characteristic of bilateral disease of the spinal cord, disseminated sclerosis, general paresis hemorrhage into the pons, and such irritating lesions of the brain as cerebral meningitis cerebral or subdural hemorrhage, and sunstroke It also occurs in the aged and in hyperopia

Unilateral Myosis: When not congenital, this may be caused by the application of a myotic, or by one of the following diseased states: A very large aneurysm exercising sufficient pressure upon the sympathetic fibers of the thorax to cause paralysis, locomotor ataxia; general paresis of the insane, or other unilateral lesion affecting the cord The same conditions may be due to unilateral cerebral lesions irritating the oculomotor nerve center.

Anisocoria (inequality in the diameter of the pupils when the eyes are at rest): This may be a congenital or a

seen as a whitish elliptical depression situated somewhat to the nasal side of the posterior pole of the orbit. The *blood vessels of the eye* (the main artery and vein) arise in the optic disk and branch out in the fundus.

Pathologically, the retina may become *colorless* in severe anemia or in ischemia, and markedly *reddened* in active or passive hyperemia Active hyperemia may be due to eyestrain or irritation. Passive hyperemia is usually due to obstruction of the retinal circulation as a result of valvular heart disease during the stage of decompensation, glaucoma, convulsions, asthma, etc.

Retinitis (inflammation of the retina): This may be due to a variety of factors, some of which cause definite pathological entities.

Retinitis may be classified as

I *Simple or Serous Retinitis:* This includes (*a*) syphilitic retinitis, (*b*) sympathetic retinitis, (*c*) retinitis from concussion. They are characterized by inflammation and engorgement of the retinal vessels often associated with edema.

II. *Parenchymatous Retinitis:* This includes (*a*) albuminuric retinitis, (*b*) diabetic retinitis, (*c*) leukemic retinitis, (d) syphilitic chorioretinitis, (*e*) hemorrhagic retinitis, (*f*) macular retinitis. These are characterized by hyperemia, engorgement of the vessels, edema, hyperplasia with involvement of the deeper structures.

(*a*) *Albuminuric retinitis* is recognized by: (1) The appearance of variously sized white or yellowish white plaques in the vicinity of the macula from which they radiate, often occupying the major portion of the retina; (2) retinal hemorrhages which are flame-shaped, linear, dotted or sheetlike, extending along the arteries, and (3) signs of neuritis or papillitis, such as indistinct outline or swelling of the optic nerve which is often streaked with diverging vessels.

(*b*) *Diabetic retinitis* closely resembles albuminuric retinitis, differing only in that the hemorrhages are smaller and there is an absence of the white radiating plaques or spots around the macula.

(*c*) *Leukemic retinitis* is characterized by the appearance of the arteries and veins. The arteries are small, pink and at times yellowish in color; the veins are large, broad and rose-red in color Opaque deposits composed of lymphocytes extend from the macula to the equator.

(*d*) *Syphilitic chorioretinitis* is first noted in the uvea, later extending to the retina; or the retina and choroid may be simultaneously affected. Both eyes may show different stages of the affection.

(*e*) *Hemorrhagic retinitis* may occur in syphilis, nephritis, cardiac disease, hypertension and arteriosclerosis. This condition is recognized by the appearance of hemorrhages in the retina and retinitis.

Hemorrhages into the retina without retinitis may occur in arteriosclerosis, anemia, septicemia, pyemia, bacterial endocarditis, purpura, hemophilia, scurvy, heart disease, strain, suffocation and trauma.

(*f*) *Macular Retinitis:* This is an inflammatory condition occurring in the macula lutea.

III. **Embolic or septic retinitis** is usually found in association with inflammation of the choroid and occurs in cerebrospinal meningitis, septicemia, trauma and infections.

IV *Retinal sclerosis* includes (*a*) *retinitis pigmentosa*, the diagnostic features of which are night blindness, di-

of the pupil when the eyelids attempt to shut against resistance.

Paradoxical Pupillary Reflex: In this the pupils dilate instead of contracting upon exposure to light or accommodation.

Consensual or Indirect Reaction: This is a condition in which the pupil on the diseased side does not react to direct light, but does react when the light is thrown into the sound eye This phenomenon is seen in diseases of the optic nerve or tract, in which neither the oculomotor nerve of the diseased side, nor its nucleus and nuclear connection with the corpora quadrigemina (and through the latter with the opposite optic tract) are involved.

Hippus: This is an alternate contraction and dilatation of the pupil which occurs under sudden exposure to light. It is often seen in normal individuals, but it occurs more frequently in hysteria, epileptic subjects, the early stages of meningitis, disseminated sclerosis, advanced paralysis, and in mania Phthisical patients occasionally display hippus, particularly at a stage when the thoracic glands are greatly enlarged, so that they cause irritation of the thoracic ganglion Alternate contraction and dilatation of the pupils is often noticed in Cheyne-Stokes respiration, the pupils dilating during the dyspneic period and contracting during apnea

The Retina (The Fundus)

The *retina* cannot be examined with the unaided eye. At times when the pupil is dilated a red glare can be seen, but no details of the nerves or vessels are visible. The retina is examined by means of the ophthalmoscope—an instrument devised by Helmholtz — the main principle of which is a concave mirror with a central aperture. The light is thrown by the mirror through the pupils upon the retina, while the examiner looks through the central aperture into the interior of the eye.

In *direct examination*, looking through the Helmholtz ophthalmoscope, or one of its modifications, or any electric ophthalmoscope, the examiner gradually approaches his own eye to the eye to be examined until the red glare of the retina is visible; he then brings his own eye in close contact with that of the patient, in order to make a detailed examination The examiner's eye and that of the patient must be of similar refractive power; if a discrepancy exists, the examiner's eye must be neutralized by one of the lenses with which the opthalmoscope is supplied The image thus obtained is designated as a "direct image."

When the *indirect method* of examination is used, the eye is illuminated from a distance of 25 to 30 cm, and a convex lens is held about 5 cm. from the eye. This lens magnifies the interior of the eye, thus presenting an "inverted image"

The interior of the eye is examined in order to determine the condition of the media, the crystalline lens and, most particularly, the retina, or the fundus, as to its color, size, condition of the blood vessels, optic cup and state of the optic nerve.

Pathological conditions of the retina are usually due to systemic disease In order to diagnose accurately retinal findings, special training in the use of the ophthalmoscope is required.

Color of the Retina: The color of the retina is usually a purplish red tint, though it varies with the complexion of the individual, being lighter in the light complexioned and darker in the brunette The *optic disk* (optic nerve entrance) is

seen as a whitish elliptical depression situated somewhat to the nasal side of the posterior pole of the orbit. The *blood vessels of the eye* (the main artery and vein) arise in the optic disk and branch out in the fundus.

Pathologically, the retina may become *colorless* in severe anemia or in ischemia, and markedly *reddened* in active or passive hyperemia. Active hyperemia may be due to eyestrain or irritation Passive hyperemia is usually due to obstruction of the retinal circulation as a result of valvular heart disease during the stage of decompensation, glaucoma, convulsions, asthma, etc.

Retinitis (inflammation of the retina): This may be due to a variety of factors, some of which cause definite pathological entities.

Retinitis may be classified as:

I. ***Simple or Serous Retinitis:*** This includes (*a*) syphilitic retinitis, (*b*) sympathetic retinitis, (*c*) retinitis from concussion They are characterized by inflammation and engorgement of the retinal vessels often associated with edema.

II. ***Parenchymatous Retinitis:*** This includes (*a*) albuminuric retinitis, (*b*) diabetic retinitis, (*c*) leukemic retinitis, (*d*) syphilitic chorioretinitis, (*e*) hemorrhagic retinitis, (*f*) macular retinitis. These are characterized by hyperemia, engorgement of the vessels, edema, hyperplasia with involvement of the deeper structures.

(*a*) *Albuminuric retinitis* is recognized by: (1) The appearance of variously sized white or yellowish white plaques in the vicinity of the macula from which they radiate, often occupying the major portion of the retina; (2) retinal hemorrhages which are flame-shaped, linear, dotted or sheetlike, extending along the arteries, and (3) signs of neuritis or papillitis, such as indistinct outline or swelling of the optic nerve which is often streaked with diverging vessels.

(*b*) *Diabetic retinitis* closely resembles albuminuric retinitis, differing only in that the hemorrhages are smaller and there is an absence of the white radiating plaques or spots around the macula.

(*c*) *Leukemic retinitis* is characterized by the appearance of the arteries and veins. The arteries are small, pink and at times yellowish in color; the veins are large, broad and rose-red in color Opaque deposits composed of lymphocytes extend from the macula to the equator.

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minution of the central vision, contraction of the visual field, occasional color blindness and a deposit of pigment along the vessels, (*b*) *retinitis proliferans* which is characterized by a proliferation of Muller's fibers with the formation of connective tissue around the optic nerve, thereby causing grave impairment of vision.

Pulsation of the Retinal Vessels: This is seen in aortic regurgitation, exophthalmic goiter and in any condition that causes throbbing of the arteries.

Tubercles in the Choroid: These are found in tuberculous meningitis and miliary tuberculosis

Choked Disks: These are found in albuminuric retinitis and tumor of the brain.

Tumors of the Retina: These may also be recognized by opthalmoscopic examination, they include melanotic sarcoma, carcinoma, glioma, etc

The Eyeball

The eyeball is examined in order to determine its tension, and its position in relation to the orbit

Exophthalmos (protrusion of the eye ball) Bilateral exophthalmos is seen in exophthalmic goiter. The eyes may appear to protrude—or perhaps do actually protrude slightly—as a result of sudden fright, or during an attack of spasmodic croup or of asthma Exophthalmos is also noted in thrombosis of the superior longitudinal sinus, in cardiac hypertrophy, particularly if due to aortic regurgitation, in laryngeal stenosis and in paralysis of the associated ocular movements One or both eyeballs may protrude because of hemorrhage in the orbit, aneurysm, exostosis or tumor of the orbit, and also because of enlarged lacrimal glands

Prominence of the Eyeballs: This occurs in near-sightedness and at times as a familial peculiarity.

Enophthalmos (recession of the eyeballs): This may be either bilateral or unilateral.

Bilateral enophthalmos may be due to absorption of fat in the orbital cavity, a



Fig 22—Paralysis of associated ocular movements

condition noted in all wasting diseases such as marasmus, pulmonary tuberculosis or the cachexia of cancer, also in long-continued febrile states, such as typhoid fever and in starvation

Unilateral enophthalmos is usually due to a lesion of the cervical sympathetic or the cranial nerves, which interferes with nutrition, causing atrophy of the orbital connective tissue, or paralysis of Müller's orbital muscles

The Orbit

The orbit may become the seat of disease, or, because of pressure or direct

extension, may produce distinct eye symptoms.

Abscess: This may be acute or chronic; it usually follows an injury. This condition may be recognized by constant pain, with redness, swelling of the eyelids, conjunctivitis, exophthalmos, and fluctuation



Fig 23—Carcinoma of eye

Fracture: This usually results from violent direct injury It may give rise to meningeal symptoms, also to inflammation and suppuration of the orbital tissue

Foreign Bodies: These may be found following injury by an explosive, such as shrapnel, or any similar accident Usually the eye itself will suffer injury at the same time, although there are cases in which the eye has entirely escaped damage. The symptoms depend upon the size of the foreign body and the extent of the injury inflicted

Periostitis: This is a painful condition which may be recognized by the presence of a tender point over one of the orbital bones.

Benign Tumors: These may give rise to pressure symptoms.

Carcinoma: This is usually secondary though it may occur as a primary growth. Glioma may be primary or secondary.

Sarcoma: This usually can be recognized by its rapid growth and the occurrence of sarcomata in other situations

Aneurysm: This occurs as a result of sudden strain, particularly in a syphilitic individual The patient can, as a rule, indicate the time when the aneurysm was formed, because of the sensation of a sudden "snap," followed by severe pain



Fig 24—Strabismus (Ebaugh)

Strabismus (Squint)

This is caused by overaction or paralysis of one or more of the eye muscles or by disease of the cranial nerves Stabismus is classified, according to its direction, into convergent (when both eyes

minution of the central vision, contraction of the visual field, occasional color blindness and a deposit of pigment along the vessels; (*b*) *retinitis proliferans* which is characterized by a proliferation of Müller's fibers with the formation of connective tissue around the optic nerve, thereby causing grave impairment of vision.

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The Orbit

The orbit may become the seat of disease, or, because of pressure or direct

ited to an astigmatic eye, it will be seen as a line, an oval, or a circle, according to the situation of the retina, but never as a dot. Horizontal, oblique, and perpendicular lines of the same breadth, arranged in one figure will appear to the astigmatic eye as lines of different dimensions Astigmatism may be simple, compound, myopic or hypermetropic, mixed or irregular.

Anisometropia: This is a condition in which one eye is more hypermetropic or myopic than its fellow.

Presbyopia ("long sight" of old age): This is a condition in which an object is partially or completely invisible at close range, but is clearly visible at a distance In general, with advancing age, the power of accommodation decreases.

Testing Visual Acuity: A standard card, usually the Snellen card, is employed in testing visual acuity The eyes are tested one at a time, the eye not in use being covered during the examination The card is placed about 20 feet distant under good illumination, and the patient is asked to read all the letters or figures which he can see distinctly The first line where the letters appear indistinct to him is considered his limit of distance, has visible acuity expressed by a fraction, in which the numerator indicates the greatest distance at which the person examined is able to read the smallest letter on the card, and the denominator the greatest distance at which a normal eye can recognize the same letter

The patient who sees at 20 feet distance the letters normally visible at that distance, has visual acuity expressed as 20/20 (normal). If he can see at 20 feet *only such letters that are normally* visible at 40 feet, then his visual acuity is only 20/40

The Ears

The External Ears: They should be examined for change in color, displacements, growths, edema and pain

Color: They may be cyanosed, pale or excessively red

Auricular Displacement: It is well to note if the two auricles are identical in the angles which they form with the sides of the head While slight differences in this respect may be due to ordinary anatomical variations, marked differences, on the other hand, are most likely due to the presence of an inflammatory condition in the ear or temporal bone of the bulging side. Marked displacement of one auricle points usually to an inflammatory process, either in the mastoid cells (acute mastoiditis) or in the wall of the fibrocartilaginous meatus (furunculosis). In acute mastoiditis the auricle is pushed outward, forward and downward The postauricular sulcus-linear depression between the auricle and side of the head is usually obliterated in suppurative inflammation, involving the tympanic cavity and mastoid cells with or without a subperiosteal abscess. When the displacement of the auricle is outward, forward and upward it usually indicates *furunculosis of the external auditory* meatus, a condition much less serious although more painful.

Growths: *Cysts* are sometimes found about the auricular region. These are *small tumors filled with clear colorless* fluid and show no inflammation. *Sebaceous cysts* are often observed in the lobule or in the skin behind it. They are commonly caused by the acumula*tion of secretion* when the sebaceous glands have been blocked for any reason. In patients subject to gout, *tophi,* de-

seem to meet—internal squint), divergent (when both eyes seem to look in different directions — external squint), and altitudinal, directed either upward or downward Divergent and convergent squint may also be either upward or downward.

Eye Signs

von Graefe's Sign: This was described both by von Graefe in Germany (1864) and Demarres in France (1856), working independently. It can be readily recognized even in relatively mild cases, but its absence does not warrant a negative diagnosis in any given individual. In directing the eye downward, the lower margin of the upper eyelid does not follow the line of vision normally, but lags behind or follows in an irregular spastic manner.

Stellwag's Sign: This is closely related to von Graefe's sign, and was first described by Stellwag in 1869. In patients suffering from marked exophthalmos there is a retraction of the upper eyelid, and at the same time the lid remains much more stationary than it does under normal conditions. There is also a marked decrease in the frequency of winking.

Moebius' Sign: In 1895 Moebius pointed out the fact that in many cases of exophthalmic goiter there is an insufficiency of convergence. If the patient is directed to look at the ceiling and then suddenly at his own nose, it will be found that only one eye will be directed toward the nose, and the other may take any direction, although it usually maintains its axis fairly parallel with the eye that is directed toward the nose. This symptom may also be elicited by having the patient fix an object with his eyes at a distance of several yards, then by gradual approach of the face a point will be reached at which one eye only will continue to fix the object, the other eye ceasing to see it. There is no definite distance from the eyes at which convergence ceases, and the distance is not even constant for the same patient at different examinations. This test is not positive in all cases of exophthalmic goiter, but can be elicited in most of these cases Several other eye signs have been described in exophthalmic goiter. For additional eye signs, see *Exophthalmic Goiter*, p. 779.

Refraction

By refraction is clinically meant the measuring of visual accuracy. Certain visual defects are correctable by glasses

Emmetropia (normal range of vision): This is a condition midway between hypermetropia and myopia. External objects produce an image which is focused accurately upon the retina.

Ametropia is a condition where the principal focus does not lie on the retina. There are three kinds of ametropia. Hypermetropia, myopia, and astigmatism.

Hypermetropia (hyperopia, farsight): In this condition, the refractive power is too weak, or the axis of the eye too short, causing the principal focus to form beyond the retina.

Myopia (nearsight): This is the condition where the refractive power is too strong or the axis of the eye too long, causing the principal focus to form in front of the retina.

Astigmatism: This is a combination of emmetropia, hypermetropia and myopia in the same eye. This condition is due to asymmetry of the meridians of the eye When a luminous dot is exhib-

media, usually mixed with pus. If the discharge is largely pus, with a slight admixture of blood, it indicates the presence of a purulent otitis media or an abscess, or it may be due to bone necrosis, cholesteatoma, or fungus infection. In fracture at the base of the skull, the presence of spinal fluid pre-



Fig 26—Testing for hearing

vents the bloody discharge from coagulating, a point sometimes of value in making a differential diagnosis

Deafness. The presence of deafness should be determined by any one of the hearing tests Deafness may be due to faulty perception Conduction deafness may be caused by blockage of the auditory canal by cerumen, foreign bodies inflammations, tumors, abscess or furunculosis, also by acute or chronic disease of the middle ear or the eustachian tube

Perception deafness is found in otosclerosis, in disease of the auditory nerve or the cochlea It may also occur in acute infectious diseases, and in tumor of the skull or the cerebellopontine angle or the auditory nerve It may result from fracture of the skull, from exposure to constant noise for an extended period, and from the use of certain drugs such as quinine or salicylates

Hearing Tests: These may be roughly carried out in three ways (1) A ticking watch is held close to the external ear, while the examiner's hand shields the patient's eye upon the same side The watch is then gradually removed from the ear until it reaches a point where the patient claims he can no longer hear it; the watch is then held at varying distances from the ear until it can be ascertained exactly at what distance hearing ceases (2) The patient is directed to turn his face toward the wall, the examiner standing from 6 to 8 feet away from him whispers certain words or numbers which the patient is asked to repeat. This procedure may be repeated with the examiner standing at different distances or raising his voice An attempt should be made to test each ear separately This can only be done by total temporary exclusion of the other ear The closure of one ear with a pledget of cotton held by the finger is not sufficient to exclude that ear from all hearing. This is especially true when testing by means of spoken or whispered words There it is necessary to employ special instruments devised for that purpose (3) By means of the audiometer, a special instrument devised for testing the acuity of hearing

Tinnitus Aurium (ringing in the ears) This may be unilateral or bilateral it may be functional as seen in the neuroses or it may be due to a lesion in the auditory apparatus associated with partial or complete deafness Tinnitus is a common complaint in middle ear disease, otosclerosis, impacted cerumen, Meniere's disease, eustachian tube obstruction, nasal obstruction, hypertension, mountain sickness, tunnel sickness, acute anemia, and drug effects, as from quinine and salicylates

posits of sodium biurate crystals, sometimes called *chalkstones*, frequently appear in the pinna margin.

Edema: A large amount of edema behind the ear may be present in both mastoiditis and furunculosis. In mastoiditis, firm pressure behind the auricular attachment directed against the bone will elicit deep-seated tenderness In furunculosis, such pressure against the bone will be painless, whereas movement of the auricle from side to side or pressure from behind the ear directed forward against the auricle causes marked pain.

Pain: When the patient complains of pain in the ear, it is well before making any instrumental examination of the drumhead to look for possible swelling and *inflammation of the external canal* Except in infants and very young children, in whom the anatomical relation between the drum membrane and external meatus is exceedingly close, manipulation of the auricle or tragus causes absolutely no pain when the inflammation is confined to the middle ear. On the other hand, very slight *movements of the auricle or tragus are* extremely painful even in the initial stages of a furuncle in the meatus.

The Meatus: This should be inspected to ascertain the presence or absence of any purulent *discharges or any obstructing* foreign matter. The *tympanic membrane* can be inspected through a speculum illuminated either by reflected light or by a small electric bulb within the speculum itself The points to note about the drumhead are its color, consistency, the presence or absence of injection or bulging, scars or perforations

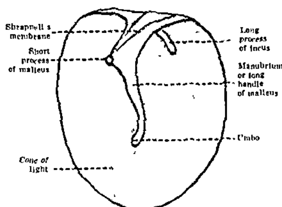


Fig 25—Ear drum, membrana tympani and structures visible

The Canal: This is examined for impactions, foreign bodies, local inflammation, furunculosis, or other lesions. The presence of fine hairs in the meatus sometimes obstructs the view of the deeper parts. In such an event the examiner after the insertion of the speculum, will apply a little vaseline to the hairy area by *means of a* cotton-tipped probe, by this means the hairs are made to adhere closely to the walls of the canal

Discharges: Discharges from the ear are of diagnostic importance. With a history of injury the appearance of blood from the external auditory meatus, if not caused by bleeding granulation tissue, indicates a fracture of the skull at the base; the blood is often mixed with cerebrospinal fluid. Blood is sometimes discharged from the ear in otitis

ceive attention from a properly qualified specialist.

Sense of Smell (SEE: p. 65): In various diseases the sense of smell may be lost (anosmia), it may become increased (hyperosmia), or it may be perverted (parosmia).

I. ***Anosmia:*** The loss of the sense of smell may be a purely local condition due to excessive dryness of the nasal mucous membrane, acute and chronic rhinitis, nasal polypi, mouth breathing, pollens or extremely offensive odors The loss of the sense of smell may also result from disease of the nasal accessory sinuses, disease or injury of the olfactory tract, bone disease in the vicinity of the olfactory bulb, basal meningitis and tumors or gumma affecting the olfactory nerve. Anosmia is a frequent complaint in neurasthenia and hysteria and is at *times found in locomotor ataxia.* Unilateral anosmia may be due to local disease of one of the nasal chambers or disease of one hemisphere of the brain.

II ***Hyperosmia:*** Increased sensitivity to odors is usually found among those who possess a hypersensitive nervous system, or among people who are susceptible to certain odors.

III ***Parosmia:*** A perverted sense of smell to the extent that the usually accepted agreeable odors are shunned as offensive, and disagreeable odors are accepted as pleasant is found in certain functional nervous derangements and in some forms of nasal catarrh. *Kakosmia* is the perception of bad odors when they are nonexistent. This is sometimes found as hallucinations in certain psychoses, head injuries and rarely in tumors of the hippocampus.

The Mouth

In studying the mouth the condition of the lips, the gums, the teeth, the buccal mucosa, the tongue, the pharynx and the larynx as well as the odor on the breath should be considered



Fig 27—Technic for inspection of teeth, gums and lips

The Lips

An examination of the lips is not complete unless they are everted so as to expose their buccal surfaces.

In anemia and wasting diseases the lips are usually pale in color; also after hemorrhage and in prolonged fevers. They may be, very dry in conditions of exhaustion and extreme thirst The lips are *fissured* in certain forms of indigestion, or after exposure to cold; fissures at the angle of the mouth (cheilitis) are found in the toothless, in vitamin B_2 deficiency, and in those who for any reason have a continual dribbling of saliva. Lip fissures in infants and young children should arouse suspicion of congenital syphilis and of some nutritional defect.

The Nose

The nose is examined as to its color, size, the condition of the nares, the presence of discharges, or of obstruction to respiration.

Color: Chronic red nose, due to dilated capillaries, is found in chronic alcoholism, acne rosacea, lupus erythematosus and persistent digestive disorders; and in such local skin conditions as pustules or boils. Superficial ulceration of the nose may be caused by tuberculous ulcer or by epithelioma; a circular, punched-out ulcer may be due to syphilis.

Size and Shape: A coarse broad nose is found in cretinism and myxedema; acromegaly causes a gradual increase in the size of the nose. A depressed sunken (saddle) nose is found in syphilis, in achondroplasia, or may be the result of an injury. A nose which appears pinched, with small nares, is indicative of the presence of hypertrophied adenoid tissue, or other chronic obstruction which causes mouth breathing. Various tumors may affect the nose, *i. e.*, angioma, carcinoma and syphilis

Playing or Dilatation of the Alae Nasi: Occurring during respiration, this is often noticed in lobar pneumonia and other pulmonary affections and cardiac disease associated with dyspnea and fever. It also occurs in neurotic individuals when under excitement.

Perforation of the Septum: The nasal septum may become perforated because of syphilis, cocaine sniffing, injury and as a result of unsuccessful septum operation

Regurgitation of Fluid through the Nares: This occurs in laryngeal diphtheria, postdiphtheritic paralysis, retropharyngeal abscess, enormously hypertrophied tonsils and in peritonsillar abscess (quinsy). Bulbar paralysis and cleft palate may also cause nasal regurgitation.

Discharges: Inoffensive *watery discharges* from the nose are noted in all cases of nasal catarrh, in the early stage of measles, in hay fever, in vasomotor rhinitis, and in acute irritation of the schneiderian membrane and the mucous lining of the nose. *Pus* may be discharged from the nares, either as a result of local infection, or from drainage of the antra or the upper sinuses *Offensive discharges* may be caused by an impacted foreign body, nasopharyngeal diphtheria, or by lupus which affects the nasal chambers. *Ozena* may be due to caries, rhinitis, or syphilitic infection; it is also found in glanders. *Epistaxis* (nosebleed) may be caused by the rupture of a blood vessel, trauma, ulceration from the presence of a foreign body, or the presence of polypi or neoplastic growths. Other causes of epistaxis are purpura hemorrhagica, scurvy, leukemia, hemophilia, aplastic and other types of severe anemia, vicarious menstruation, telangiectasis and excessive high blood pressure.

Nasal Obstruction: This may be due to polypi, a deviated septum, hypertrophied turbinates, hypertrophic rhinitis, acute coryza, hay fever, nasal diphtheria, or foreign bodies in the nose "Snuffles" is a pathognomonic sign of hereditary *syphilis.*

The nasal cavities and their contents are examined with the aid of a nasal speculum by reflected or direct light. A complete examination of the nose and sinuses requires the use of special apparatus, and training beyond the attainment of the ordinary practitioner. *All pathologic nasal conditions should re-*

ceive attention from a properly qualified specialist.

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Herpes (vesicles): Commonly known as *"cold sores,"* these often appear in malaria, pneumonia, typhoid fever, acute coryza and many other febrile diseases

Eczema: *This usually occurs on both* lips. They are dry, fissured, bleed easily and are often covered with crusts.



Fig 28—Harelip and cleft palate. A congenital malformation

Chancre: The initial lesion of syphilis not infrequently makes its appearance upon the lip. *It* is characterized by an indurated base and gives off a thin secretion, and is usually accompanied by *enlargement of the submaxillary glands* In considering the nature of a "sore" upon the lip which suggests chancre, the history should be minutely scrutinized; numerous instances are on record of innocent extragenital syphilitic infection which has taken place upon the lip A classic example is *that* cited by Schamberg,[1] where a number of young girls were thus infected by playing "kissing games" at a social gathering where one of the male guests was in the active infective stage of syphilis

Condyloma Latum: The mucous patch characteristic of syphilis com*monly appears on the lips in the form of* a flattened, strictly delimited area, coated with gray exudate, and is usually found *at the angle of the mouth.*

Epithelioma: This is one of the most malignant forms of skin cancer Its early identification is of the utmost importance In the initial stages there is a possibility of confusing it with chancre. Trauma, especially long-con*tinued trauma,* as from constant'y holding a pipestem at a certain spot between the lips, or continual irritation by a jagged tooth, or badly fitted artificial denture, plays an important part in the etiology of epithelioma of the lip. In the differential diagnosis the history is of signal importance. Chancre is commoner in



Fig 29—Chancre of lip

young subjects, while epithelioma in any location seldom appears before the age of 40, though a sufficient number of exceptions to this rule have occurred to render the diagnosis still more difficult. The appearance of early lip epithelioma is

[1] Schamberg, J F. *An Epidemic of Chancre of the Lip from Kissing* Jour. Amer. Med Assoc, lvii; 783, Sept 21, 1911.

similar to the common cold sore, a painless crack, fissure or other break in the continuity of the mucous membrane of the lower lip (less than 5 per cent of all cases occurring upon the upper lip. The lesion is covered by a crust or "scab,"



Fig 30—Condyloma latum (mucous patch).

which leaves a raw surface when removed, and immediately re-forms without any tendency to healing. The ulcer gradually becomes indurated at the edges and increases in size slowly, seldom giving the patient pain or inconvenience until it is well advanced. Later, involvement of the cervical and submaxillary glands will take place. Any lip lesion which does not heal promptly, especially in a patient of middle age or over, or where no luetic history is obtained, should be carefully watched and vigorous measures instituted as soon as the need for them becomes apparent, as practically all hope of cure lies in early recognition

Carcinoma: This is usually secondary to carcinoma in its immediate vicinity. In rare instances primary carcinoma of the lip may be manifested.

Tuberculous Ulcer: This is not uncommonly seen among the chronic actively tuberculous. The ulcer is usually situated at the inner portion of the lip close to the angle of the mouth. The diagnosis may be verified by pathological examination.

Angioneurotic Edema: This may occur upon either lip as a sudden painless disfiguring swelling, resembling a bee sting or mosquito sting. The swelling may disappear in a comparatively short time on the administration of epinephrine.

Harelip: This is a congenital deformity of the upper lip It may be unilateral and affect a small portion of the lip, the entire lip or extend to the hard palate, or it may be bilateral



Fig. 31—Epithelioma of the lower lip.

The Gums

Color: The color of the gums has important diagnostic significance. In all forms of anemia the gums show marked pallor. If they display a bluish line at

the teeth edges it is indicative of lead poisoning; a greenish line in the same location may indicate copper poisoning; in scurvy, the gums are of a purplish color; a bluish red tint is indicative of mercurial stomatitis. A red line on the gums of a young adult



Fig 32—Carcinoma of lip.

probably indicates gingivitis, though it may be due to one of several possible affections of the teeth, *i. e.,* to pyorrhea, or lack of proper hygiene of the mouth. In a child it is often an early sign of scurvy. As temporary hyperemia may confuse the examiner in determining the presence of a definite line of color upon the gums, it is well to insert a toothpick or a piece of white paper between the gum margins, thus raising them slightly; if the discoloration remains after the gum margin has been raised, it indicates a true discoloration, rather than a temporary hyperemia.

Spongy Gums: This and ulceration upon the gums, are often found in gingivitis, particularly when the teeth have been ill-kept; also when there are large deposits of tartar upon the teeth, or in the presence of gangrenous stomatitis, scurvy, poisoning by phosphorus, by mercurial or by radioactive substances; and in some constitutional diseases like diabetes, leukemia, tuberculosis and certain digestive disturbances, and in Vincent's angina.

Stomatitis: This is an inflammation of the buccal mucous membrane. It may affect the entire mouth or only the gums, the cheeks, the tongue or any local portion of the mouth. The lesions may be erythematous, macular, papular, pustular, or ulcerative. It may occur as the result of local or general infection or of trauma.

Vincent's Angina (trench mouth, necrotic gingivitis): The gums are ulcerated and necrotic, a white line of



Fig 33—Vincent's angina affecting the gums.

necrotic tissue covers the tooth margins and extends downwards, often spreading to the lips, cheeks, tongue and pharynx. The teeth are covered by the necrotic exudate, and the mouth odor is extremely fetid.

The Teeth

Eruption of the Teeth: It is important for every practitioner of medicine to be familiar with the approximate time when both the deciduous and permanent teeth should appear.

It is exceedingly necessary to know when a deciduous tooth either should be or may be extracted.

Deciduous Teeth: The commonest order of eruption is:

Two central incisors in the lower jaw, at six to nine months.

The four upper incisors appearing in pairs from 8 to 12 months, those in the center coming in before the lateral pair.

Two lower lateral incisors 12 to 14 months.

Four anterior molars from 12 to 15 months.

Four canines from 18 months to 2 years.

Four posterior molars between the second and third years.

A child one year old should, therefore, have six teeth; at a year and a half old it should have 12 teeth; at two years 16 teeth; and between two and a half and three years, 20 teeth.

When the deciduous teeth have remained in position some years their apices begin to be absorbed to make room for the subjacent development of the permanent teeth. Such absorption begins from two and a half to three years before the permanent teeth erupt, and continues until the whole of the root has been absorbed, when the tooth is, or should be, shed When the permanent teeth erupt their roots are not fully formed, and the apical foramina are large and patent, absorption of toxins, bacteria and dangerous drugs is very likely to occur, if they gain access to, or are applied to, the pulp during the stage of open apices, either in deciduous or permanent teeth. The ages at which the apices are "closed" are from two and a half years to three years after the eruption (except the canine teeth which are nearly complete at eruption).

Permanent Teeth: The permanent teeth come in as follows:

First molars at six years of age.

Incisors at seven to eight years.

Bicuspids at nine to ten years

Canines at 12 to 14 years.

Second molars at 12 to 15 years.

Third molars ("wisdom teeth"), 17 to 25 years.

Rickets, cretinism, severe anemia and hereditary syphilis usually *delay dentition.*

Irregular Dentition: The upper teeth may erupt before the lower in cretinism, rickets and malnutrition

Inspection of the Teeth: It is imperative that every general physical examination should include a careful inspection of the teeth. It is now universally recognized that a host of ailments, formerly attributed to a wide variety of causes, owe their origin to some focal infection in the mouth, most commonly an alveolar or periapical abscess.

This writer sounds a note of warning against the present tendency to overemphasize the importance of oral sepsis to the exclusion of everything else. The general examination should include a careful survey of the condition of the teeth, their general appearance as to health and cleanliness, whether they are decayed or loose, and if they present any noticeable abnormalities. It is also important to observe whether the patient is wearing any kind of artificial denture.

Complete examination of the teeth cannot be made without resorting to

radiography, and this aid should always be called in if the examiner has any reason to suspect the presence of infective foci

Decay and Malformation: *Caries:* Decay and loosening of the teeth is usually found in badly nourished and early life, while *pitted teeth* may be the result of severe stomatitis during childhood. Both conditions result from hypoplasia of the enamel

Hutchinson's Teeth: *This is a* designation applied to the notched and narrow-edged permanent incisors often

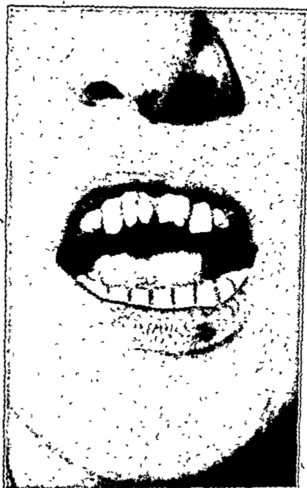


Fig. 34—Hutchinson's teeth.

feeble children, and in adults who do not carry out proper hygiene of the mouth, likewise in the presence of diabetes, rickets, scurvy, pyorrhea alveolaris, chronic phosphorus poisoning, and mercurial stomatitis.

Furrows: In an adult these may usually be attributed to severe illness in

characteristic of congenital syphilis; the tooth is short and narrow, smaller at the cutting edge than at the root; there is usually a single narrow and discolored notch at the cutting edge. The teeth are as a rule irregular and set wide apart. While Hutchinson's teeth are regarded as indicative of congenital

syphilis, they are not invariably of such origin.

Sordes (filth): This is the collection of dark brown foul matter upon the teeth which is sometimes seen in conditions of prostration, or in pneumonia, typhoid fever, and whenever the typhoid state is present. It consists of a mixture of food, epithelial matter and micro-organisms.

The Tongue

The tongue is to be studied not only for local disease, but also for signs of systemic affections.

Size: *Macroglossia* (large tongue): This is usually congenital, though it may occur later in life as a result of inflammation of the lymphatics, glossitis, Ludwig's angina, actinomycosis, acromegaly, or myxedema. Localized swelling of the tongue may be caused by such tumors as gumma or carcinoma, by cysts, fibroma, by foot-and-mouth disease, and by local trauma.

Microglossia (small tongue): This may occur because the tongue has become somewhat atrophied as a result of severe hemorrhage. It is seen also in an advanced state of emaciation, in anemia, or in convalescence from typhoid fever. Disease of the hypoglossal nerve, bulbar palsy and cerebral syphilis may cause a slight atrophy of the tongue. Local diminution in size may result from a gumma or the extensive scar formation following a deep ulcer or other injury.

Tongue Lesions: *Scars:* These have a diagnostic significance because they may occur as a result of injury, such as accidental biting of the tongue, or biting during an epileptic seizure, restless sleep, careless mastication, or from a blow upon the chin while the tongue was protruded. Bulbar palsy sometimes causes ulceration of the tongue which results in the formation of scars.

Fissures: These are at times found in perfectly healthy individuals; the cause of this phenomenon is obscure, and occasionally it may be due to vitamin B deficiency; it does not in any way interfere with function. Very deep and inflamed fissures may be due to dissecting glossitis, a frequent result of syphilitic infection, and to leukoplakia A fissured tongue may be caused by a broken tooth, and it may result from chronic dysentery, diabetes mellitus, and chronic hepatic disease.



Fig 35—Simple ulcer of the tongue.

Tumors: These may be benign or malignant (SEE: Fig. 37, p 195).

Benign tumors are fibroma, neurofibroma, lipoma, fibrolipoma; keloid cysts, lingual thyroid, angiomata and papilloma. These are usually free from pain, do not cause metastasis and do not ulcerate. The lymphatics at the angles of jaw and of the neck are not affected.

Malignant tumors are carcinoma and sarcoma. They usually ulcerate, cause severe pain and give rise to metastasis and enlargement of the nearby lymph glands

Ulcers: These may result from syphilis, tuberculosis, or stomatitis, the last

two being painful. Very shallow ulcers, with a red glazed surface, appear in chronic superficial glossitis. Multiple ulcers, grayish, indolent and of stellate shape, when associated with enlarged cervical glands, are probably tuberculous in origin. In syphilis also we find multiple ulcers, but the glands are rarely enlarged, multiple small ulcers produced by vesicles are found in smallpox, chickenpox, measles, empyema, herpes, pemphigus and eczema of the tongue.



Fig 36—Tuberculous ulcerations of the tongue.

A *single ulcer with an indurated base* and associated with enlarged cervical glands may sometimes be the result of stomatitis, but it is more likely to be an epithelioma, and for this reason all such lesions upon the tongue should have the closest scrutiny, and their exact nature carefully ascertained If an epithelioma has reached the stage of cervical gland-enlargement before being brought to the examiner's attention, the patient has but a small chance of cure or even relief of symptoms. The diagnosis must be based on the history, therefore inquiry should be made whether this spot has been subjected to long-continued trauma, such as smoker's burn or jagged or decaying teeth, and as to its failure to heal after a reasonable length of time following its appearance, its indolence and gradual extension, and the age of the patient Epithelioma is essentially a disease of middle age.

Other causes of a single ulcer upon the tongue are gumma, abrasion by a jagged tooth, or possibly an accidental biting.

Leukoplakia (psoriasis linguae) This is a small, thick, elevated patch upon the tongue, of irregular size and shape. It is not ulcerated, but is smooth and white, feeling hard to the touch It is often a forerunner to epithelioma

Geographic Tongue: This is so named because of the presence of ring-shaped patches of denuded epithelium, which spread at the edge while healing at the center These patches eventually coalesce, forming irregular areas with curving outlines. It is sometimes called "mappy tongue" This is of indefinite pathologic significance.

Coating and Color of Tongue: Normally the tongue is of a clear red color, with a slight coating, but occasionally it may be markedly coated in healthy subjects, particularly in smokers and mouth breathers.

In severe anemia the tongue is *pale,* and appears smaller than the normal A *red* tongue is seen in inflammations such as glossitis, stomatitis, and certain infectious diseases; in cyanosis it is of a *bluish tint, yellow* in jaundice, and *dark red* in conditions of great prostration. In Addison's disease it is marked

by *purple, dark brown,* and *black* deposits.

Strawberry or *mulberry tongue* is pathognomonic of scarlet fever, being so called because of the peculiar redness of the tongue and its raised papillae.

Glossophytia, black tongue, is a condition in which the tongue has a black coat upon the dorsum which is due to



Fig 37—Tumor of the tongue

the presence of microphytes. It may also be due to vitamin B_2 deficiency. *Black tongue* in the dog is analogous to pellagra in the human The tongue may be stained *brown* by the use of chocolate, licorice, tobacco, laudanum or rhubarb, while iron, bismuth and charcoal cause a *black stain* upon it

Staining and *superficial necrosis* of the tongue may be due to the ingestion of corrosive substances; hydrochloric, sulphuric and nitric acids will stain it *yellow*, it will be turned *white* by the action of ammonia, corrosive sublimate, carbolic, and oxalic acids; caustic alkalies, fruits and wine will cause it to turn *red*

There are a number of conditions in which the tongue assumes a shape, discoloration, and dryness sufficiently definite to have diagnostic importance. Among such are the thin white furrowing of the tongue (often noted in perfect health especially in smokers and mouth breathers) characteristic of nasopharyngeal catarrh, caries of the teeth, mild gastric catarrh, and mild febrile conditions. A flabby, swollen, indented tongue, uniformly covered with a yellow pasty "fur," particularly on arising in the morning, is often seen in those who smoke much, or use alcohol freely; it is also found in patients suffering from gastritis and nephritis, and in long-continued fevers in which the temperature does not rise very high. A tongue that appears narrow, the center covered with a thick rough fur, the median fissure deepened, and the tip and edges red and denuded, is characteristic of the typhoid state and is usually seen in typhoid fever. A dry, brown, fissured tongue which is protruded slowly and tremulously, and not withdrawn until the patient is told to do so, is often met with in those who are critically ill, a desquamating tongue, protruded and withdrawn in the same manner, indicates a similar condition. A dry red ("beefy") tongue is seen in low fevers, associated with severe toxemia, dysentery, hepatic abscess and chronic intestinal catarrh; when the tongue becomes moist and the coating gradually disappears, it is an indication that the patient is recovering A tongue which is gray and flabby, with red irregular spots, so that it has a worm-eaten leafy appearance, is often seen in disease of the buccal mucosa occurring in children. Unilateral furring of the tongue is often the result of irritation of the second or third division of the trigeminal nerve; it is also noted in unilateral paralysis of the tongue. Localized small furring may be caused by a roughened tooth, by local

inflammation, or by an inflamed tonsil. A grayish coating of the tongue in adults, or a white coating in children may be due to thrush, in which case other parts of the buccal mucosa will be similarly affected. A small, pale, smooth tongue is characteristic of pernicious anemia.

Manner of Protrusion. Very sick patients will protrude the tongue slowly and incompletely; it will be put out with hesitation and not immediately withdrawn unless the patient is told to do so. This is especially noted in advanced cases of typhoid fever, or any condition presenting the typhoid state, and in general toxemia; the tongue will be tremulous in the early stages of typhoid and in meningitis, in chorea, it is thrust out with a sudden peculiar jerk, and immediately withdrawn

General tremor of the tongue is noted in alcoholism, asthenia, Graves' disorder, and in bulbar palsy; in the last mentioned it is accompanied by fibrillary twitchings. Deviation of the tongue toward the paralyzed side may occur in hemiplegia when the face is affected When the tongue deviates toward the sound side, it indicates a lesion in the medulla.

Spasm of the tongue occurs in stuttering, also in multiple sclerosis, general paresis and melancholia.

Impediment in the power of protrusion of the tongue frequently occurs in paresis, diphtheritic palsy, progressive muscular atrophy and some forms of hemiplegia. The tongue cannot be protruded by patients who have spasms of the muscles of mastication, general convulsions, tetanus ("lockjaw"), or any painful condition of the muscles which prevents the mouth from being opened, such as trismus neonatorum, strychnine poisoning and, at times, hysteria and epilepsy. Inability to protrude the tongue may also be the result of irritating lesions in the region of the fifth nerve, or of chronic spasms of the muscles of the jaw, when the teeth are "chattering" from cold or mental excitement, or during a chill. This condition occasionally occurs also as the result of some irritation of the teeth and jaw.

Taste: There are four primary taste sensations perceived by the tongue Sweet, bitter, sour and salt, a combination of any two or more of these primary taste sensations may be recognized Complete loss of the sense of taste may result from bilateral disease of the chorda tympani nerve and from disease of the gustatory fibers of the glossopharyngeal nerves Partial loss of taste may result from disease of the gustatory fibers or of the chorda tympani on one side.

Technic for Testing Taste Sensations: Small quantities of quinine solution, vinegar or hydrochloric acid solution, syrup and sodium chloride may be placed in succession upon the protruded tongue, the patient being asked to point to one of four cards with the proper answer, "sweet, sour, bitter, salt," etc

Gustatory Agnosia: Loss or impairment of the sensation of taste may be due to an unhealthy condition of the lingual mucous membrane, involving the "taste buds," the end organs of the gustatory nerve fibers. Agnosia may be present when the tongue is heavily coated, or when it has been in contact with some irritating substance. Agnosia is often an associated symptom of acute coryza. Aside from the conditions already named, the loss of taste sensation often occurs in basal meningitis, when tumors are present, or when an injury to the head has taken place. The sensation of taste is usually lessened when the tongue is dry.

Parageusia: Perversion of the sense of taste may result from the administration of such drugs as potassium iodide, the bromides, or tartar emetic. "Bad taste" is usually one of the complaints in gastroduodenal catarrh, jaundice, and other conditions which produce a "furred" tongue. Perversion of the taste sensation is present in certain functional nerve derangements, such as hysteria, or the hallucinations of the insane

Lingual Pain: This is found in the presence of local lesions of the tongue, in glossitis, fissures, malignancy and in pernicious anemia; also in macrocytic and microcytic anemia, in sprue, pellagra, and vitamin B deficiency

The Palate

The palate should be examined to ascertain its color, and the presence or absence of rashes, *inflammation or paralysis* A rash is often visible upon the palate in measles, giving an appearance of minute circumscribed vesicles (Koplik spots, also seen on the cheek) Mucous patches are seen as a manifestation of secondary syphilis, and vesicles arranged in circles upon the soft palate and the pharyngeal wall, which are painful, are an indication of herpes of the throat

Swelling of the Uvula: This is often noticed in inflammatory conditions of the pharynx and tonsil. The uvula may *also become edematous in nephritis, in* severe anemia, in angioneurotic edema or in grave cases of general debility Membranous exudate upon the uvula *extending to the palate is usually caused* by diphtheria and Vincent's angina. Bloody extravasation of the uvula is noted in purpura hemorrhagica and certain other cases of hemorrhagic diathesis.

Paralysis of the Soft Palate: This may result from diphtheria, neuritis, bulbar paralysis, tumor at the base of the brain, basal meningitis and vertebral caries.

Anesthesia of Soft or Hard Palate: This may result from disease which involves the second division of the fifth nerve.

The Tonsils

A careful inspection of the tonsils is an essential part of every physical examination. It should be noted carefully whether they are hypertrophied or inflamed or covered by any exudate. The condition of the crypts should also be scrutinized. Enlarged and inflamed tonsils may be due to an acute inflammation, such as follicular tonsillitis, influenza, pharyngitis, scarlet fever, diphtheria, acute mononucleosis, agranulocytic angina, and other infections.

Hypertrophy of the Tonsils: This usually becomes chronic in early childhood. The examiner should bear in mind the fact that a focus of infection may be hidden in the tonsil, even when to all appearances upon a superficial examination the tonsil seems healthy. As in the case of the teeth, an infectious focus in this location may be the cause of constitutional disturbances in a remote part of the body, a possibility which must always be considered

Exudates: A whitish-gray punctate exudate *which occupies the crypts or* the surface of the tonsil may be due to *follicular tonsillitis;* a gray and confluent exudate, spreading to the pillars, the fauces, the soft palate and other neighboring structures, is probably caused by *diphtheritic infection.* Such a membrane may be removed, but it will leave a bleeding surface. Deep circular ulcers which present a gray surface while the remaining portions of the ton-

sil appear normal, result from *syphilitic infection.* In *tuberculosis of the larynx* or *pharynx,* irregular grayish ulcers will often be visible upon the tonsils, the exudate frequently having the appearance of frog's spawn. In an elderly person, deep spreading ulcers upon an enlarged tonsil, which give off an offensive exudate, should arouse a suspicion of *malignancy.* A *heavy grayish exudate upon the tonsils alone or also upon the gums may be caused by *Vincent's angina.* A healing throat after tonsillectomy causes a thick grayish exudate.

The Pharynx

The pharynx is examined as to inflammatory conditions, exudates, and ulcers.

Redness: This may be caused by *acute pharyngitis often seen in naso*-pharyngeal catarrh, influenza, tonsillitis, scarlet fever, Vincent's angina, diphtheria, and the early stage of measles; it may also be caused by irritations produced by food that is too hot or too cold.

Ulcerations: These may be caused by syphilis, tuberculosis, diphtheria, cancer, and lupus. Small ulcers may also result from chronic pharyngitis, and similar ulcers are sometimes found in the terminal stages of typhoid fever Bulging forward of the posterior pharyngeal wall indicates the existence of a *retropharyngeal abscess, or an abscess* due to caries of the cervical vertebrae

Anesthesia: This takes place when conditions exist which affect the glossopharyngeal or pneumogastric nerves. It is also seen in diphtheria, bulbar paralysis and neuritis. *Globus hystericus,* imaginary "lump in the throat," is frequently witnessed in hysteria, and is said to be due to a functional disturbance of the ninth nerve. Acute gastritis and esophagismus will often cause patients to complain of the sensation of a lump in the throat.

Spasm: This is usually a functional disorder. It may be present in hydrophobia, tetanus, or strychnine poisoning; it is also found *in neurotic and hysterical* individuals.

Paralysis: *This is* caused by a lesion which involves the ninth and tenth cranial nerves, it may also be seen in bulbar paralysis, Landry's palsy (acute ascending spinal paralysis), basal meningitis, cranial tumors or aneurysm, and sometimes in neuritis.

Dysphagia (pain, or difficulty in swallowing): This may be caused by disease of the tongue, swelling of the tonsils, disease affecting the muscles of *the neck, and by any inflammatory con*dition of the mouth, tongue, pharynx, or larynx due to ulcers or other reaction to irritation. Dysphagia may also be caused by ulceration, stricture, or by the presence of a tumor of the esophagus which constricts the lumen, or by an aneurysm.

The Breath

The odor of the breath will vary according to the kind of food or drugs which may have been ingested. Such odors as those of orange, pineapple, onions, or garlic, are familiar examples of foods which impart a distinctive odor to the breath. An odor like that of peach kernels is imparted to the breath by hydrocyanic acid; a garlicky odor by overdoses of arsenic. Opium, ether, chloroform, and alcohol have each their characteristic odor which needs no description. An unpleasant foul odor of the *breath is often caused* by stomatitis, caries of the teeth, necrosis of the jaw.

tonsillitis, diphtheria, abscess and gangrene of the lung, and by fetid bronchitis, bronchiectasis and pyothorax. Various forms of gastrointestinal disturbances associated with indigestion will impart an unpleasant odor to the breath. A "strong odor" on the breath may also be due to pharyngolaryngeal catarrh, or may be caused by various disturbances in the nose, or its communicating sinuses.

A *urinous odor* of the breath is indicative of uremia, while a *sweetish odor,* similar to that of overripe apples, is often found in diabetes mellitus, particularly during the coma stage. An *odor* like that of the breath of *carnivorous* animals is often noted in those who are critically ill and who are suffering from marked acidosis or alkalosis.

The Neck

The neck is examined by inspection, palpation and at times also by auscultation

Inspection: The color of the skin, visible glands, visible pulsations and enlargements are thus studied

Palpation: The glands are studied as to their mobility, consistency and size Pulsations are studied as to their origin, whether arterial or venous

Technic for Palpating Glands of the Neck: For the posterior cervical chain of glands, the patient's head is slightly bent forward and the examiner runs the fingers of both hands along the trapezius and occipitofrontalis muscles The anterior chain of glands are studied in a similar manner, preferably with one hand, the thumb being on one side of the neck and the index and middle fingers on the other The patient's chin is tilted upward while the examiner's hand is slid up and down along the side of the neck. In order to determine the position of the trachea, the thumb is placed between the anterior belly of the sternocleidomastoid muscle immediately above the suprasternal notch. The amount of space on one side of the trachea as palpated with the thumb is compared with the space on the opposite side A narrowing indicates deviation of the trachea toward that side. (For technic "for the detection of arterial and venous pulsations," SEE p. 524)

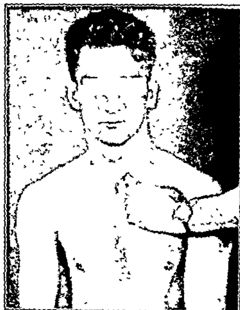


Fig 38—Palpating trachea to note its position and proximity to the sternocleido mastoid muscle

Two Methods of Palpating the Thyroid Gland: (1) The index finger and thumb of one hand or the index fingers of both hands gently grasp the anterior portion of the neck near the anterior bellies of the sternocleidomastoid muscles immediately above the clavicles; if any mass is felt the patient is asked to swallow The thyroid gland when enlarged may be felt moving up and down during deglutition.

2. The patient tilts the chin upwards, the examiner gently presses the index and middle fingers of his hand against the lateral aspect of the trachea, thus pushing it aside and the thumb of the same hand palpates for the thyroid gland during the act of deglutition

A substernal thyroid may be outlined only by percussion and the x-rays.

Tracheal Tugging: This may be elicited by having the patient sit upright, head somewhat lowered. The examiner stands behind the patient and hooks the first phalanx of each index finger above the suprasternal notch, thus supporting the cricoid cartilage. A steady rhythmical pull or tug, synchronous with the heartbeat, when felt by the palpating fingers, indicates a tracheal tug The sign is often present in aneurysm of the aortic arch

Tracheal tugging may at times be found in simple, nonaneurysmal dilatation of the aorta; in mediastinal tumors adhering to both the trachea and aortic arch, and in other inflammatory conditions of the mediastinum involving the aortic arch.

The neck muscles are studied as to rigidity and tenderness. Touching or feeling the muscles will usually elicit tenderness when present. *Rigidity* of the neck muscles is determined by grasping the prominent muscles between the thumb and fingers and noting their degree of elasticity Rigidity of the neck as a whole is determined by the examiner slipping his hand under the occiput and an attempt is made to raise the head off the pillow. In the presence of rigidity, instead of the head flexing, the entire body is lifted.

Auscultation: This is employed for the determination of a venous hum or a murmur.

The neck is also studied as to its mobility, the condition of its glands, the presence of existing pulsations in excess of those normally present, and for the presence of tender areas and rashes.

If the neck is more freely movable than normal, it indicates that a fracture of some of the cervical vertebrae has occurred, or a complete relaxation of the muscles from loss of nerve control has taken place. Any disease of the neck which affects its mobility is apt to take the form of rigidity, which may be slight or marked

Rigidity of the Neck: This may be caused by disease of the cervical vertebrae; by *spasms of the cervical muscles*; inflammatory conditions of the throat; inflamed cervical glands; furuncles or carbuncles, meningitis; tetanus and strychnine poisoning *Torticollis,* wryneck, may be congenital or acquired as a result of scars, cervical rib, disease of the cervical vertebrae, adenitis, tonsillitis, rheumatism, retropharyngeal abscess, enlarged cervical glands, injury to the sternocleidomastoid muscle, and cerebellar tumor.

The Glands

Normally, the *thyroid gland* is barely visible, pathologically it may be enlarged, either slightly, or to a marked degree. Moderate enlargement if not due to Graves' disease does not give rise to any symptoms, and may often be observed in adolescent girls at the time of puberty; sometimes also after childbirth or during the menopause.

Cystic goiter is the usual cause of a greatly enlarged thyroid The gland may be greatly hypertrophied, yet give rise to no other symptoms than those of pressure.

Parenchymatous goiter causes enlargement with few symptoms.

Exophthalmic goiter (Graves' disease) is a disease in which the thyroid gland may become enlarged and present a definite group of symptoms (syndrome) including exophthalmos, tachy-

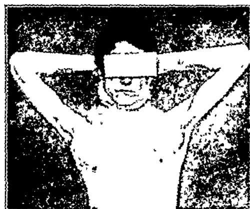


Fig 39—Hodgkin's disease

cardia, tremor, and at times mental disturbances The eye signs are discussed on pp. 182 and 779

An abnormal enlargement of the thyroid which pulsates is due to vascular changes (struma vasculosa). This at times has to be differentiated from dilated aorta or aortic aneurysm occupying the suprasternal notch (SEE: p. 531)

Atrophied thyroid is recognized by a peculiar depression in the location of the gland; a condition found in myxedema and cretinism

Glandular hypertrophy occurs in various suppurative diseases, especially in childhood For diagnostic purposes the glands should be studied as to their position, size and consistency, *i e.*, hard or fluctuating. It should also be noted whether the swelling is of an acute or chronic type.

At the angle of the jaw, behind the ramus, the glands in the upper part of the neck will often become acutely swollen in diphtheria, tonsillitis, scarlet fever, German measles and other exanthemata; also in erysipelas, glanders or retropharyngeal abscess, and occasionally in caries of the teeth.

Chronic enlargement of the cervical glands may be found in the following diseases: In *tuberculosis* the glands are large, matted and show a tendency to suppuration. In *syphilis* they are bilaterally affected, small and hard, and do not suppurate In *Hodgkin's disease* the glands are large, isolated and nonsuppurating, and are associated with glandular hypertrophy in other parts of the



Fig 40—Benign submaxillary tumor.

body. In *lymphatic leukemia* the cervical glands may be greatly enlarged; they are soft, freely movable under the skin, not tender to touch, and do not suppurate. The overlying skin is not inflamed. In this disease nearly all the superficial

lymph nodes become enlarged. In *lymphosarcoma* the cervical glands grow rapidly and form large masses. They are not freely movable underneath the skin, are often tender to the touch and have a tendency to infiltrate the adjacent structures. In *status lymphaticus*



Fig 41—Branchial cyst

the cervical and axillary glands are palpable; they seldom become very large This condition is found in childhood and is accompanied by the general classical appearance of the child, *i e*, fat, flabby child, large tonsils, enlarged thymus gland and hypoplasia of the heart and blood vessels

Mumps (specific parotitis) presents an acute swelling, which appears just in front and immediately behind the ear The carotid lymph nodes are sometimes enlarged as a result of an inflammatory condition of the pharynx and of the skin of the face.

In *gumma* the swelling is at first hard, the overlying skin becomes red; later the mass softens and breaks down, forming a punched-out ulcer.

The *posterior cervical glands*, particularly those lying under the upper extremities of the trapezius and occipito-frontalis muscles, often become enlarged as a result of eczema of the scalp, pediculosis capitis, or of syphilis.

The group of *superficial cervical glands* above the clavicle is often hypertrophied as a result of cutaneous disease upon the face, neck, or external ear

The *glands of the submaxillary group* may be enlarged because of caries of the teeth, stomatitis, tonsillitis, mumps, syphilis, and cancer of the tongue or lower lip



Fig 42—Actinomycosis

Enlargement of the glands immediately above the left clavicle is often found in malignancy of the abdominal

viscera, above the right clavicle in intrathoracic malignancy.

Among the other causes for glandular enlargement, the following should be borne in mind:

Carbuncle usually occupies the back of the neck, causing inflammation and



Fig 43—Diffuse lipoma of the neck

induration which eventually undergoes necrosis

In *cellulitis* the skin is swollen, red and hardened

Ludwig's angina causes swelling and induration affecting the undersurface of the chin.

Superficial abscess is characterized by a fluctuating mass localized to one side or posteriorly

Cysts, thyroglossal and branchial, are hard and painless. They are formed either on the midline or near the left sternocleidomastoid muscle, and contain mucus or dermoid material

Actinomycosis usually involves the upper part of neck and lower jaw; often starts as a lumpy swelling in the region of the parotid and submaxillary glands The skin involved is red, elevated and covered with small nodules which eventually break down.

Anthrax (malignant pustule) occurs upon the back of the neck, face and hands. The pustule breaks early and forms a large, indurated, painful, black or purplish mass with a central depression. The surrounding skin becomes edematous.

Mikulicz's disease causes a brawny, noninflammatory swelling of the parotid, submaxillary, sublingual and lacrimal glands. It is usually symmetrical

Submaxillary sialadenitis may affect one or both submaxillary glands, usu-



Fig 44—Aneurysm of neck

ally in children; it is moderately tender and painful; as a rule, it results from blocking of the salivary duct.

Infectious mononucleosis (glandular fever) has a sudden onset, moderate temperature, some laryngitis; the tonsils or

gums may be inflamed, and often there is a mild papular or macular rash on the body. The superficial and often the deep lymph glands of the neck, axilla, groin or mesentery become enlarged There is a moderate leukocytosis with a



Fig 45—Compressing a pulsating vessel in the neck in order to note if pulsation is above or below point of compression, and to observe if vessel fills from above or below.

great increase in the number of lymphocytes and a decrease in the number of polymorphonuclear leukocytes. The heterophile antibody test is positive in high dilutions.

Lipoma may be simple or diffuse; may affect a portion of the neck or surround it collar fashion; it is painless and not tender to touch.

Tularemia (Rabbit Fever). In the oculoglandular type the regional lymph glands of the neck enlarge early.

Aneurysms of the innominate or subclavian arteries are recognized by their expansile pulsation, thrill and *bruit.*

Pulsations of the Neck

These may be either arterial or venous. Arterial pulsations are usually found in aortic regurgitation, arteriosclerosis, aneurysm of the ascending aorta, exophthalmic goiter and extreme emaciation; they are also often noted after violent exercise.

Venous pulsations may be caused by tricuspid regurgitation, cardiac decompensation, Stokes-Adams syndrome, auricular fibrillation, patent foramen ovale with mitral regurgitation, and anemia. Pulsations in the episternal notch may be due to aneurysm of the aorta, exophthalmic goiter, anemia, and may occur often in the aged when great emaciation has taken place.



Fig 46—Pellagra.

Method of Differentiating Arterial from Venous Pulsation: A pulsating artery is not as easily compressed as a pulsating vein. When a pulsating vessel in the neck is compressed (with

one finger) midway between the angle of the jaw and the clavicle, and pulsation is noted below the point of compression and none above it, it is an indication of arterial pulsation. But if fullness and pulsation is noted above the point of compression and none below it, it is an indication of venous pulsation, because the veins fill from above downward, while the arteries fill from below upward

Engorgement of the Jugular Veins: The jugular veins are normally more prominent during expiration than during inspiration Pathologically they may become prominent during cardiac decompensation, presenting a positive venous pulse. In obstruction of the superior vena cava by mediastinal tumor, aneurysm, chronic adhesive pericarditis, enlarged bronchial glands, large pericardial effusion and retropharyngeal abscess, one or both jugulars may become distended. In bronchial asthma, in chronic emphysema and in pertussis during a severe paróxysm of coughing, because of strain upon the pulmonary circulation, right-sided cardiac dilatation and venous engorgement often result.



Fig 47—Tuberculosis cutis

Tenderness of the Neck

Tenderness of the neck is usually present when the neck muscles are inflamed, either because of muscle injury or reflexly as a result of inflamed glands,

bone injury, cerebral disease or some form of inflammatory skin disease.

Localized tenderness of the neck is found in acute tonsillitis, diphtheria and German measles (over the lymph glands and at the angles of the jaw); in peritonsillar abscess and after tonsillectomy (over the lateral muscles of the neck); in Pott's disease, dislocation or fracture of a vertebra (over the affected spine); in diaphragmatic pleurisy and at times in pericarditis (along the trapezius muscles), in aneurysm of the aortic arch (over the left sternocleidomastoid muscle) The presence of a cervical rib may at times be demonstrated by eliciting pain on pressure over the inner part of the clavicle, the pain usually radiating down the arm.

Rashes Upon the Neck

The neck, like any other portion of the body, may be the seat of such skin eruptions as eczema, psoriasis, acne vulgaris, tinea versicolor, tinea circinata, erythema multiforma, the various syphilodermis, etc. In addition to those mentioned, several rashes have a predilection for the skin of the nape of the neck, among these are boils, carbuncles, venekeloid, scrofuloderma, neurodermite, and lichenification

SECTION 6

The Thorax and Respiratory System

CHAPTER IX

Topographic and Regional Anatomy of the Thorax

The thorax or chest is a bony case, covered externally by muscles, fat and skin, and lined internally by pleura The upper boundary is formed by the clavicles, and the lower boundary by the twelfth ribs. The dividing line between the thoracic cavity and the abdomen is the diaphragm, a musculomembranous partition, the insertion of which corresponds to the following levels: *Anteriorly,* the sixth rib; *laterally,* the eighth rib; and *posteriorly,* the tenth rib All the organs within the confines of the ribs, if above the diaphragm, i e, the lungs, heart, etc, are considered as being *intrathoracic,* while those below the diaphragm, though partially costal, i e, the liver, spleen, kidneys and a portion of the stomach, are considered *intraabdominal.*

Devoid of its fleshy covering, the thorax is conical in shape It is customary to describe it as possessing an anterior, a posterior and two lateral aspects, an anteroposterior diameter — which gives it its *depth* — and a *transverse* diameter — which imparts breadth The anteroposterior diameter of a normal thorax is usually three-fourths of its transverse The thorax is practically formed by the ribs, these bones being united posteriorly in the median line to the spinal column The seven upper ribs are reinforced posteriorly by the scapulae, while anteriorly, they are joined by their costal cartilages to the sternum, which permits an up-and-down movement of the ribs with the extension of the sternum This upward movement of the ribs and extension of the sternum causes *chest expansion.*

In order to facilitate the study of the thoracic cavity contents, we utilize certain anatomical landmarks situated on the anterior and posterior aspects of the chest wall, and lay down arbitrary lines having a fixed anatomic starting point

Anatomic Landmarks and Rib Counting

The important anatomic landmarks of the chest are the ribs, the clavicles, the sternum, the mammary glands and nipples, the scapulae, and the spinal column.

The Ribs

The ribs are the most important of the bony landmarks utilized for studying the lungs, heart and other thoracic organs; it is, therefore, very important to be able to localize the various ribs when a physical examination of the chest is made

First Ribs: Each first rib is covered by its respective clavicle, the space immediately below is the first intercostal space *Each intercostal space is,* therefore, *below its corresponding rib;* the second intercostal space below the second rib, the third intercostal space below the third rib, and so on The first, second, and third intercostal spaces are wider than the rest; all *intercostal spaces are wider anteriorly than they are laterally, and are narrowest posteriorly.*

Second Ribs: The second ribs are the easiest to locate. They correspond anteriorly to a horizontal ridge of bone known as the *angle of Louis* or *Louis' angle,* which is formed by the junction of the *manubrium* and the *gladiolus.* It

is also the landmark for the bifurcation of the trachea. The pulmonary artery bifurcates near the left second rib, at its sternal end; the beginning of the aortic arch is near the second rib, at its sternal end; the upper border of the scapula corresponds posteriorly to the second rib.

Third Ribs: *Posteriorly, the spines* of the scapulae are on a level with the third ribs.

Fourth Ribs: In lean males, or young girls, the nipples are on a level with the fourth ribs.

Fifth Ribs: The fifth ribs correspond to the lower external border of each pectoralis major muscle.

Sixth Ribs: When the arms are raised in a horizontal line, the sixth ribs correspond to the highest visible digitation of the serratus magnus. *A horizontal line drawn through the nipple* will be on a plane with the *sixth rib or* the sixth intercostal space in the midaxillary line

*Seventh Ribs: Anteriorly, the sev*enth ribs are on a level with the sternoxiphoid articulation; laterally, they correspond to the second lowest digitation of the serratus magnus muscle. Posteriorly, the lower angles of the scapulae rest on the seventh ribs, when the arms are held in the normal anatomical position, and on the eighth ribs when the arms are held perpendicular to the chest.

Eighth Ribs: The last visible digitation of the serratus magnus lies over the eighth ribs.

Ninth Ribs: A line encircling the body on a level with the first lumbar vertebrae will meet the ninth ribs in the midclavicular line.

Tenth Ribs: *The tenth ribs are the* last of the fixed ribs and can, as a rule, be felt at the midclavicular line.

Eleventh and Twelfth Ribs: The eleventh and twelfth ribs are the "floating ribs" and can be readily palpated in most lean individuals

Though each rib has a distinct landmark of its own, the most accurate way of counting ribs is by locating *Louis' angle,* which is formed by the junction *of the manubrium and gladiolus* and corresponds to the level of the second ribs. From this point the other ribs are easily counted by allowing the index finger to palpate each rib and intercostal space successively. When counting laterally and posteriorly, the general course of the ribs must be borne in mind. Anteriorly, they run a nearly horizontal course; laterally, they slope upward; while posteriorly, they are almost oblique. This sloping position of the ribs causes them to be much lower at their *sternal articulation than they are at the* vertebral column The chondrosternal articulation of the third ribs is on a level with the body of the sixth dorsal *vertebra. Below this, to the seventh rib* inclusive, there is a difference of four ribs between the posterior and anterior articulations. Thus, a *horizontal line en*circling the body at a level with the fourth ribs anteriorly will fall upon the eighth ribs at their spinal articulation, and so on. In other words, adding the number four to the number of the rib in front (third to seventh inclusive) will give the number of the rib at the corresponding level near the spine.

Clavicles

The "collarbones," one on each side of the sternum, occupy the uppermost position of the chest framework, and act as a dividing line between the neck and the thorax. The subclavian artery passes under the clavicle near its sternal

articulation. The center of this bone is utilized as the starting point for the mid-clavicular line.

Sternum

The sternum or breastbone divides the anterior aspect of the chest into a right and a left half. It articulates on either side with the cartilages of the *seven upper ribs*.

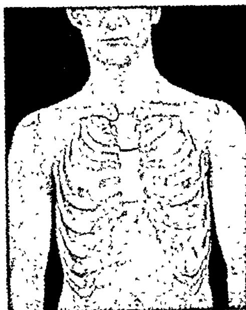


Fig 1—The normal thorax, anatomical relations of clavicles, ribs, sternum, and nipples

The *suprasternal notch* is the depression at the top of the sternum between the sternal ends of the clavicle; it is on a plane with the cartilaginous disk between the second and third dorsal vertebrae. At the junction of the manubrium and gladiolus—or about one and one-half inches below the suprasternal notch—a transversely projecting ridge can nearly always be felt which marks *Louis' angle* (angulus Ludovici).

Louis' angle has already been emphasized as a very important landmark because it corresponds to the second ribs anteriorly, to the disk of the fourth dorsal vertebra posteriorly, to the bifurcation of the trachea; it also marks the bifurcation of the pulmonary artery and the beginning of the aortic arch; it is the point where the lungs approach the sternum on either side. The extreme upper part of the left auricular appendage of the heart reaches the level of the angle of Louis.

The *epigastric angle* is formed by the converging and coalescing cartilages of the right and left lower ribs, which join the sternum Normally it approaches a right angle, becoming slightly obtuse during deep inspiration, and somewhat acute during expiration The apex of the epigastric angle is on a level with the disk between the tenth and eleventh dorsal vertebrae.

The *sternoxiphoid articulation* forms the apex of the epigastric angle and, as pointed out before, corresponds to the seventh sternochondral articulation and the cartilaginous disk between the ninth and tenth dorsal vertebrae A nipple-like projection, or a circular depression, or often both, mark this junction.

Mammary Glands

The mammary glands are situated on either side of the sternum between the third and sixth ribs or intercostal spaces in males and young girls. The position of the breasts in the adult female varies considerably, depending upon the pendulous condition of these organs. The *mammilla* or nipple is located in the center of the mammary gland, and lies approximately over the fourth rib in the nonpendulous breast. A longitudinal line passing through the center of the clavicle often corresponds to the center of the nipples.

Scapulae or Shoulder Blades

These are situated on either side of the spinal column. The superior border lies over the second rib posteriorly. The spine of the scapula is on a level with the third rib. It corresponds *to the dividing* line between the upper and lower

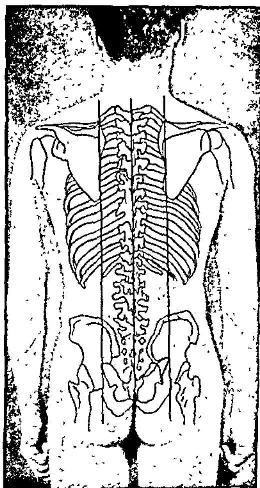


Fig. 2—Relation of the scapulae to the ribs.

lobes of the lung, and marks the upper part of the *great lung fissure.* The inferior angle of this bone lies over the upper part of the seventh rib.

Spinal Column

The spinal column is centrally situated on the posterior aspect of the chest and abdomen. The dorsal vertebrae are easily recognized as lying between the seventh cervical and *first lumbar vertebrae.* The spine of the seventh cervical vertebrae corresponds to the extreme *apex of the lung. The first rib lies immediately* below this spine. The dorsal spinous process may be utilized for rib counting This is best accomplished by having the patient bend forward, the convexity of the spine thus obtained causing the spinous processes to separate and stand out more prominently. These prominences may be still further emphasized by rubbing a towel up and down the spine, which will cause a bright red spot to mark the tip of each process, thus facilitating the counting, which should begin from the vertebral prominence or the seventh cervical spine. Because of their downward projection, the spinous processes correspond with their *next numbered rib; that is, the third* dorsal spine corresponds with the fourth rib, the fourth spine with the fifth rib, *and so on, excepting the first and the* two last ribs, which correspond with their respectively numbered vertebral spines.

The spinal vertebrae may be further utilized as landmarks for the following structures.[1]

Cervical:

First: Level of hard palate.

Second: Level of free edge of upper teeth.

Second and Third: Superior cervical ganglion of sympathetic.

Fourth: Hyoid bone.

Fifth: Middle cervical ganglion.

Sixth: Cricord cartilage, beginning trachea.

[1] *Modified from Morris.*

Seventh: Inferior cervical ganglion —apex of lungs.

Thoracic:

First: Apices of lungs.

Second: Episternal notch (interarticular cartilage).

Third: Lowest limit of superior mediastinum. Origin of greater lung fissure.

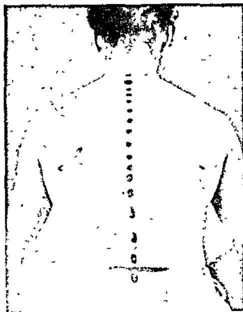


Fig 3—The spinous processes are indicated by dots which may be utilized for rib counting The second or the heavier upper dot represents the seventh cervical spine The curved lines indicate the lower angles of the scapulae. The lower horizontal line is a continuation of the iliac line and is utilized as a landmark for spinal puncture. It represents the intervertebral disk between the second and third lumbar vertebrae

Fourth: Angle of Louis, bifurcation of trachea, bifurcation of pulmonary artery, beginning of aortic arch, root of the lungs

Fifth: Termination of third piece of aortic arch; root of lungs

Fifth to Eighth: The heart.

Sixth: Pulmonary and aortic valves.

Seventh: Mitral orifice.

Eighth: Tricuspid orifice.

Ninth: Lower level of manubrium; opening in diaphragm for inferior vena cava; upper limit of spleen

Tenth: Opening in diaphragm for esophagus, level of tip of xiphoid cartilage; posterior lower limit of lung; liver comes to the surface posteriorly; cardiac orifice of stomach

Eleventh: Lower border of spleen; suprarenal capsules.

Twelfth: Lowest part of pleura; aorta passes through diaphragm (upper border); celiac axis (lower border), pylorus; upper border of kidney.

Lumbar:

First: Pancreas, pelvis of kidney; renal arteries (ending).

Second: Spinal cord ends at junction of first and second; third section of duodenum; receptaculum chyli.

Third: Lower border of kidney; umbilicus on level with third interarticular cartilage

Fourth: Bifurcation of abdominal aorta, highest part of iliac crest.

Fifth: Commencement of superior vena cava.

Sacral: *First and Second:* No important landmarks.

Third: End of first section of rectum; lower limit of spinal membranes; coccyx (tip); end of second section of rectum.

For spinal nerves and their distribution, see page 822

Arbitrary Lines

A number of horizontal and vertical lines may be drawn upon the surface of the thorax, so as to divide it into various regions or spaces. The object of this is to visualize the thoracic organs in their relation to one another and to facilitate localization and description of the pathologic lesions occurring in them.

Horizontal Lines: ***Anterior Aspect:***

I. The *cricoclavicular line* is drawn from the acromial end of the clavicle upward and inward, following the upper border of the trapezius muscle It crosses the neck in a horizontal line at the level of the cricoid cartilages, then descends along the border of the opposite trapezius muscles until it reaches the acromial end of the clavicle on that side.

II. The *clavicular line* crosses the anterior chest wall at the level of the clavicles.

III. The *third costal line* is drawn at the level of the lower border of the third ribs, running from one anterior axillary line to the other.

IV The *sixth costal line* is drawn at the level of the lower border of the sixth ribs, and runs from one posterior axillary to the other, thus not only marking the inferior border of the mammary region, but also acting as the dividing line between the superior and inferior axillary regions.

Posterior Aspect:

I. The *scapular spinal lines* are horizontal lines drawn upon the posterior aspect of the chest at the level of the scapular spines (third dorsal vertebra). Each line has its starting point at the midscapular line, thence running outward.

II. The *infrascapular line* is drawn across the posterior aspect of the chest at the level of the inferior angles of the scapulae (seventh dorsal vertebra).

III. The *twelfth dorsal line* is drawn as a slightly convex line (convexity upward) from the tips of the twelfth ribs across the posterior surface of the chest, on a level with the twelfth dorsal vertebra

Vertical Lines: On the *anterior aspect* of the chest seven vertical lines may be drawn, three on each side of the sternum, and one through its center.

The *lateral aspect* has three such lines on each side.

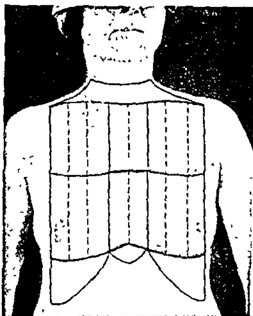


Fig 4—Arbitrary lines on the anterior aspect of the chest

The *posterior aspect* has three vertical lines, one corresponding to the spine, and one on each lateral half of the posterior aspect of the chest, passing through the lower angle of the scapula of that side.

Anterior Aspect:

I. The *mesosternal* (midsternal) *line* runs through the middle of the sternum

II. The *right and left sternal lines* correspond to the right and left margins of the sternum.

III. The *midclavicular or mammillary lines,* one on each lateral half of the chest, have for their starting point the center of the clavicle. This line often corresponds to the center of the

nipple, and terminates at the level of the sixth rib.

IV. The *two parasternal lines,* each occupies a position midway between the right or left sternal and the midclavicular line on its respective side

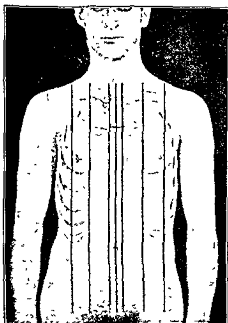


Fig 5—Vertical lines, anterior aspect of the chest

Laterally on Each Side:

I. The *anterior axillary line* is a line dropped downward from the point where the pectoralis major leaves the chest when the arm is held in a horizontal position (anterior axillary fold).

II. The *midaxillary* (mesoaxillary) *line* is drawn from the middle of the axillary space, or midway between the anterior axillary and the posterior axillary line

III The *posterior axillary line* runs through a point where the latissimus dorsi leaves the chest when the arm is in the horizontal position (posterior axillary fold)

Posteriorly:

I. The *mesospinal line* runs vertically along the vertebral spine.

II. *Scapular lines,* each passes vertically through the inferior angle of its respective scapula.

Regions of the Chest and Their Contents

Anterior Aspect

The *anterior aspect* of the chest is divided into 13 regions; two supraclavicular, two clavicular, two infraclavicular, two mammary, two inframammary, one suprasternal, one superior sternal and one inferior sternal



Fig 6—Vertical lines, lateral aspect of the chest

The Supraclavicular Regions: These are triangular spaces, each situated above its respective clavicle (right and left) Their boundaries are formed:

Anteriorly: By the sternomastoid muscle.

Posterolaterally: By the trapezius muscle (or cricoclavicular line).

Inferiorly: By the upper edge of the clavicle.

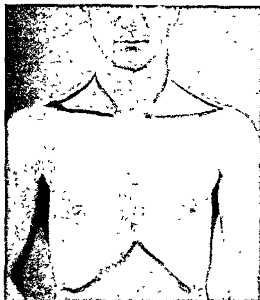


Fig. 7—Supraclavicular spaces and epigastric angle.

The floor is formed by the first rib: ***Contents:*** The same on both sides

1. Apex of the lung and its investing pleura
2. Subclavian artery
3. Carotid artery and vein.
4. Termination of the external jugular vein
5. Lymph nodes

The apex of the left lung usually rises somewhat higher than that of the right

The Clavicular Regions: These correspond to the width of the inner two-thirds of the clavicle

Contents: Lung and pleura on both sides, and in addition:

RIGHT SIDE

1. Bifurcation of the innominate artery near the sternal articulation
2. Subclavian artery a little external to the above.

LEFT SIDE

1. Carotid and subclavian arteries (deep).
2. Termination of thoracic duct.

The Infraclavicular Regions: There is one on either side of the upper portion of the sternum. Their boundaries are formed:

Superiorly: By the undersurface of the clavicle (clavicular line).

Inferiorly: By the lower border of the third rib (third costal line).

Externally: By the anterior axillary line

Internally: By the right or left edge of sternum respectively (sternal lines)

Contents:

RIGHT SIDE

1. Upper lobe of right lung and its pleura
2. Right primary bronchus (behind second articulation)
3. *Superior vena cava*
4. Part of the aortic arch. The two latter are close to sternal border.
5. Right pulmonary artery

LEFT SIDE

1. Upper lobe of the left lung and its pleura
2. Left primary bronchus (below the second costal cartilage)
3. Left pulmonary artery (edge of sternum immediately below the second sternocostal articulation).
4. Left auricle (second interspace covered by lung).

The Mammary Regions: There is one on each side of the sternum They are bounded:

Superiorly: By the lower border of third rib (third costal line).

Inferiorly: By the lower border of sixth rib (sixth costal line).

Externally: By the anterior axillary line, on each lateral half.

Internally: By right or left sternal lines respectively.

Contents:

RIGHT SIDE

1. Lung (lower part of upper lobe, the middle and a small portion of the lower lobes).
2. Pleura
3. Greater and lesser fissures of the right lung.

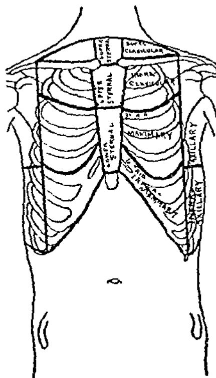


Fig 8—Regions of the anterior and lateral surfaces of the chest.

4. Right border of the heart (portions of the right auricle and ventricle covered by lung)
5. Diaphragm (during expiration it often rises as high as the fourth rib or intercostal space)
6. Dome of the liver (under the diaphragm)

LEFT SIDE

1. Lung (part of upper lobe including the lingula at fourth rib—the quadrilateral space, and a small portion of the base of the lower lobe).
2. Pleura.
3. Great fissure
4. Right auricle and ventricle; extreme border of the left ventricle and cardiac apex (fifth intercostal space ½ inch to the right of midclavicular line) or 2½ inches to the left of the midsternal line. Pericardium
5. Diaphragm.
6. Cardiac end of stomach.

The Inframammary or Hypochrondriac Regions: These are conical in shape, with their bases upward and the apex pointing downward

The *superior boundary* is formed by the lower border of the sixth rib (sixth costal line).

Inferior boundary is formed by the lower border of the tenth rib

External boundary is formed by the anterior axillary line.

Internal boundary is formed by the edges of the converging and coalescing ribs (costal arch).

Contents:

RIGHT SIDE

1. Lowest portion of the middle and lower lobes of the lung (particularly during inspiration) and pleura (complementary sinus).
2. Diaphragm
3. Liver.

LEFT SIDE

1. Lowest portion of the base of the anterior and posterior lobes of the lung (during deep inspiration).
2. Diaphragm
3. Complementary sinus (pleura).
4. The tip of the left lobe of the liver.
5. *Cardiac end of the stomach.*
6. Spleen (particularly when enlarged).

The Suprasternal Region: This is situated above the sternum and includes the suprasternal notch; it is bounded on either side by the sternomastoid muscle.

Contents: Normally it contains chiefly the trachea; pathologically it may be encroached upon by dilatation of the

aorta or an aneurysm of the aortic arch, or by an enlarged thyroid gland.

The Superior Sternal Region (upper sternal region): This has for its *upper boundary* the top of the sternum

Lower boundary is formed by a line corresponding with the lower boundary

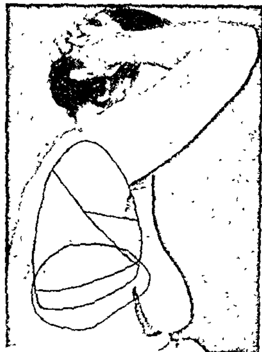


Fig 9—Regions and contents of right lateral aspect of chest

of the infraclavicular region (third rib, or third costal line)

Lateral boundaries are the right and left sternal lines

Contents:

1. Bifurcation of the trachea (near upper border of second rib)
2. Both primary bronchi
3. Inner edges of right and left lungs and their pleura, below second rib
4. Ascending and transverse arch of the aorta—in second intercostal space
5. Innominate artery, near second right costal cartilage
6. Esophagus
7. Superior vena cava
8. Left innominate vein
9. Pulmonary artery and its valve.
10. Appendix of the right auricle.
11. Thymus gland (in children).
12. Lymph nodes

The Inferior Sternal Region (lower sternal region): This corresponds to the remainder of the sternum

Contents:

1. Inner edges of both lungs
2. Small portion of upper and inner edge of left lung (above fourth rib)
3. Base of right ventricle
4. Part of right auricle
5. Part of left ventricle with the origin of the aorta (behind).



Fig 10—Regions and contents of left lateral aspect of chest.

6. Lower portion (origin) of the pulmonary artery
7. Pulmonary, aortic, mitral and tricuspid valves
8. Inferior vena cava
9. Pericardial attachment of the diaphragm
10. Left lobe of the liver.

Lateral Aspect

The lateral aspect of the chest is formed above by the armpit, below by the margin of the false ribs and on either side by the anterior and posterior axillary lines. This surface is arbitrarily divided into two regions, *viz* the *axillary* and *infraaxillary* regions.

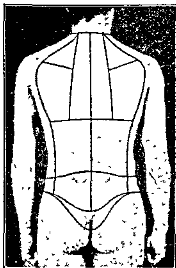


Fig 11—Arbitrary lines in regions of posterior aspect of chest

The Axillary Regions (right and left) · These are bounded

Superiorly: By the apex of the axilla

Inferiorly: By the sixth rib (sixth costal line).

Laterally: By the anterior and posterior axillary lines

Contents:

RIGHT SIDE

1 Upper, middle and inferior lobes of the right lung and its pleura
2 Greater and lesser fissures of the lung
3 Bronchi and branches (deep)

LEFT SIDE

1 Upper and lower lobes of the left lung and its pleura
2 Primary fissure.
3 Bronchi and branches (deep).

The Infraaxillary Regions (right and left): These are bounded:

Superiorly: By the sixth rib (sixth costal line).

Inferiorly: By the lower margins of the false ribs.

Laterally: By the anterior and posterior axillary lines

Contents:

RIGHT SIDE

1 Lung and pleura (base at eighth rib)
2 Diaphragm (eighth rib)
3 Liver (right lobe).

LEFT SIDE

1 Lung and pleura (to eighth rib)
2. Diaphragm
3. Spleen (ninth to eleventh ribs)
4. Stomach (portion of cardiac end at the lower level of this region).

Trube's Semilunar Space: This is bounded:

Superiorly: By the lower border of the left lung.

Inferiorly: By the spleen

Internally: By the left lobe of liver

Externally: By the costal margins

Contents: Fundus of stomach and splenic flexure (when distended).

Posterior Aspect

The *posterior aspect* of the chest may be conveniently divided into seven regions. They are a right and left suprascapular, right and left scapular, one interscapular, and a right and left infrascapular The spinal column acts as the dividing line between the right and left regions.

The Suprascapular Regions: These correspond to the supraspinous fossae and are triangular in shape The boundaries are

Superiorly and Externally: By the trapezius muscle.

Inferiorly: By the spine of the scapula

aorta or an aneurysm of the aortic arch, or by an enlarged thyroid gland.

The Superior Sternal Region (upper sternal region): This has for its *upper boundary* the top of the sternum.

Lower boundary is formed by a line corresponding with the lower boundary

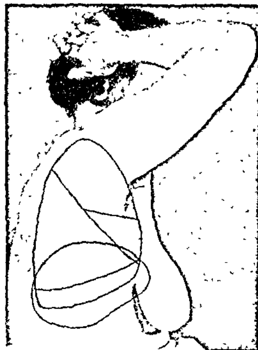


Fig 9—Regions and contents of right lateral aspect of chest.

of the infraclavicular region (third rib, or third costal line)

Lateral boundaries are the right and left sternal lines

Contents:

1. Bifurcation of the trachea (near upper border of second rib)
2. Both primary bronchi
3. Inner edges of right and left lungs and their pleura, below second rib
4. Ascending and transverse arch of the aorta—in second intercostal space.
5. Innominate artery, near second right costal cartilage
6. Esophagus
7. Superior vena cava
8. Left innominate vein.
9. Pulmonary artery and its valve.
10. Appendix of the right auricle.
11. Thymus gland (in children).
12. Lymph nodes

The Inferior Sternal Region (lower sternal region): This corresponds to the remainder of the sternum.

Contents:

1. Inner edges of both lungs
2. Small portion of upper and inner edge of left lung (above fourth rib)
3. Base of right ventricle
4. Part of right auricle
5. Part of left ventricle with the origin of the aorta (behind).



Fig 10—Regions and contents of left lateral aspect of chest

6. Lower portion (origin) of the pulmonary artery
7. Pulmonary, aortic, mitral and tricuspid valves.
8. Inferior vena cava
9. Pericardial attachment of the diaphragm
10. Left lobe of the liver.

outward and downward to the sixth rib to become the lower border, thus forming the *quadrilateral space* or notch which exposes the right ventricle of the heart.

Hilum: Each lung is attached to the inner wall of the thorax at the level of the fourth and fifth dorsal vertebrae

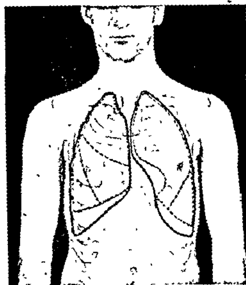


Fig 12—Anatomic position of the lungs in relation to the ribs, sternum and pleura

This attachment is known as the *root of the lung* or *hilum*, and is composed of a main bronchus, pulmonary vessels and lymphatics, held together by connective tissue and enveloped by the pleura. The lower boundaries of the lungs are·

Anteriorly, the sixth rib (in the midclavicular line).

Laterally, eighth rib (the midaxillary line)

Posteriorly, tenth rib (at the scapular line)

The variations of the number of rib or intercostal space of the lower lung limits are not only due to the lungs being lower posteriorly than they are laterally or anteriorly, but also to the peculiar slant of the ribs. It will be remembered that anteriorly the sixth rib is on a level with the posterior portion of the tenth rib.

While the general outline of both lungs is similar, there still exists sufficient dissimilarity in their structure to warrant differentiation.

Right Lung

1 Apex extends ½ to ¾ of an inch above the clavicle.
2 Has three lobes
3. Has two fissures
4. Shorter and thicker than the left.
5. Weighs about 630 Gm (21 ounces) in the male, and 540 Gm. (18 ounces) in the female

Left Lung

1. Apex extends 1 to 1¼ inches above the clavicle
2 Has two lobes
3. Has one fissure.
4 Longer and thinner than the right
5 Weighs about 570 Gm (19 ounces) in the male, and 480 Gm (16 ounces) in the female

The weight of the lungs varies with the amount of blood and serous fluid they contain As a rule larger people have larger lungs. The lungs in the male weigh about $\frac{1}{37}$th of the body's weight; while in the female they are $\frac{1}{43}$rd of body's weight

The Quadrilateral Space: This is formed by the oblique and downward recession of the anterior edge of the left lung, from the fourth sternochondral articulation to the parasternal line, at the fifth rib it again turns toward the sternum, thence slightly inward and downward to the sixth rib to form the lower border

The Lobes of the Lungs: They may thus be outlined:

Internally: By the spinal column.

Contents: Same on both sides:

1. Apex of the lung and pleura.
2. The only portion of the upper lobe found posteriorly.

The Scapular Regions: These correspond to the infraspinous fossae and are bounded:

Superiorly: By the spine of the scapula (third rib) (scapular spinal line).

Inferiorly: By the inferior angle of the scapula (seventh rib).

Posteriorly: By the vertebral border of the scapula.

Anteriorly: By the posterior axillary line.

Contents: Similar on both sides. They contain lung tissue and the greater fissure of the lung

The Interscapular Region: This is situated between the vertebral borders of the scapulae and the second to seventh ribs (the length of the scapulae).

Contents:

1. Lung tissue, hili of lungs
2. Trachea (in front of spinal column from sixth cervical to its bifurcation at the fourth dorsal vertebra into the primary bronchi).
3. Bronchial glands (clustered near the bifurcation of the trachea).
4. Descending aorta (to the left of the vertebral column).
5. Thoracic duct (to the left of the vertebral column).
6. Esophagus (to the left of the vertebral column).

The Infra- or Subscapular Regions: These are bounded:

Superiorly: By a line uniting the inferior angles of the scapulae.

Inferiorly: By the edge of the thorax (twelfth dorsal line).

Internally: By the midspinal line.

Externally: By the posterior axillary line.

Contents:

RIGHT SIDE

1. Lung and pleura
2. Diaphragm
3. Liver.
4. Kidney and adrenal gland.

LEFT SIDE

1. Lung and pleura (base at tenth rib)
2. Aorta
3. Diaphragm
4. Kidney and adrenal gland.
5. Intestines.
6. Spleen
7. Thoracic duct

The Lungs

The lungs are covered by the pleurae and are suspended by their respective roots, hanging freely in the thoracic cavity. They occupy all of that space except the mediastinum and the quadrilateral free space. The apices rise three-quarters to one and one-quarter inches above the first rib; the anterior borders of the lungs follow an oblique course downward from the apex to the level of the second rib, where they meet the sternum. From this point they pass perpendicularly downward near the median line, in apposition to one another, to the level of the fourth rib From this level the anterior border of each lung varies

The *right lung* continues downward along the sternum and slightly outward to the sixth rib, where it turns sharply to the right and becomes the lower anterior border.

The *left lung* recedes at the fourth rib in a somewhat downward course to a little beyond the parasternal line, then comes slightly forward to the fifth rib. forming the "lingula," and finally curves

then passes obliquely downward and forward, reaching the midaxillary line at the seventh intercostal space, and terminates with the lower border of the lung, at the sixth rib in the midclavicular line.

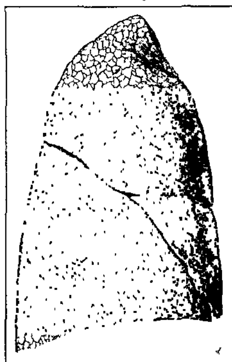


Fig 15—Right lung, showing the greater fissure, the lesser fissure, and the upper, middle and lower lobes

The *right lung* is divided into three lobes by two fissures, the *greater* and *lesser* fissures The *greater fissure* of the right lung runs a course similar to that of the left lung, commencing and terminating at the same points, *i e.*, third rib posteriorly, fourth intercostal space laterally and sixth rib anteriorly It separates the upper and middle lobes from the lower.

The *lesser fissure* branches off from the *greater* at the level of the fourth rib, near the outer border of the scapula. It runs a nearly horizontal course forward, terminating anteriorly a little below the fourth rib, thus dividing the anterior lobe of the right lung into an upper and middle lobe.

The lower surfaces of the lungs are *concave, conforming to the shape of the* diaphragm which they cap. The diaphragm reaches to the level of the fourth rib on the right side, and to the fifth rib on the left side, though the anteroinferior border of both lungs reaches the sixth rib.

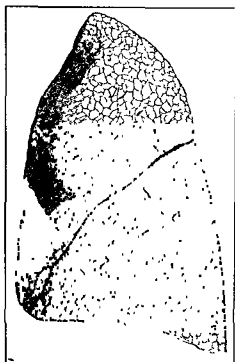


Fig 16—Left lung, showing the greater fissure, and upper and lower lobes.

The Pleura

The pleura is a serous membrane which forms a sac for each lung and lines the thoracic cavity. The two layers of the pleura are spoken of as the *visceral* and the *parietal* layer. The *visceral pleura* closely invests the lungs

Anteriorly:

RIGHT LUNG

1 Upper lobe, apex to fourth rib.
2 Middle lobe, fourth to sixth rib.
3 Lower lobe, fifth to sixth rib near the *anterior axillary line*

LEFT LUNG

1. Upper lobe, apex to sixth rib.
2. *Lower lobe, fifth to sixth rib near the* anterior axillary line.

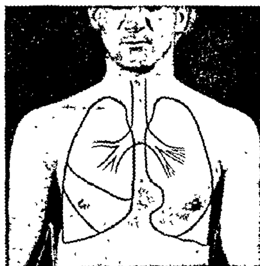


Fig 13—Fissures of the lungs, and quadrilateral space

Laterally (at midaxillary line) ·

RIGHT LUNG

1 Upper lobe, apex to fourth intercostal space
2 Middle lobe, fourth to fifth intercostal space.
3. Lower lobe, fifth intercostal space to eighth rib.

LEFT LUNG

1 Upper lobe apex to fourth intercostal space
2 Lower lobe, fourth intercostal space to eighth rib

Posteriorly:

RIGHT LUNG

1 Upper lobe, apex to third rib or fourth dorsal spine, near spinal articulation (spine of scapula).
2. Lower lobe, third to tenth rib.

LEFT LUNG

1. Upper lobe, apex to third rib or fourth dorsal spine, near spinal articulation.
2 Lower lobe, third rib to tenth intercostal space.

It should be borne in mind that the relative position of lungs and ribs varies greatly with the act of respiration During inspiration the lungs fill out, so that the apex rises higher and the base descends; at the same time the ribs become elevated During expiration the bases of the lungs rise and the ribs descend. Therefore, during inspiration—particularly when forced—the bases of the lungs may extend one or two rib levels lower, while during forced expiration the lung level may be one or two rib levels higher than when the lungs are in repose

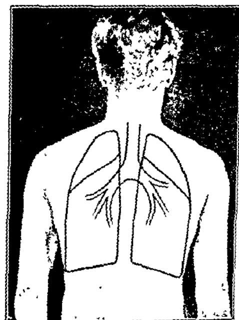


Fig 14—Fissures of the lungs.

Fissures of the Lungs: The *left lung* is divided into an upper and lower lobe by one fissure, called the *greater* or *primary fissure.* It commences at the vertebral border of the lung at the level of the third rib (spine of the scapula),

At its insertion it is on a level with the fourth intercostal space or fifth rib. The right half rises somewhat higher than the left. The upper surface of the diaphragm is in relation to the base of both lungs, the right ventricle and the pericardium. The lower surface is in relation to the liver, the suprarenal bodies, the kidneys, the spleen and the cardiac end of the stomach.

The diaphragm has three large *foramina* which permit the passing of:

1. The inferior vena cava at the level of the ninth dorsal vertebra.
2. The esophagus (to the left of the midline) on a level with the body of the tenth dorsal vertebra.
3. The aorta, vena azygos major and thoracic duct at the level of the twelfth dorsal vertebra.

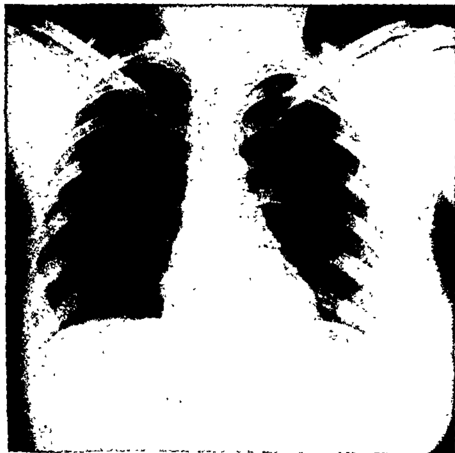


Fig 17—X-ray appearance of normal chest (Anteroposterior view.)

and dips into and lines the interlobar fissures. It fits the lung snugly at the upper part, but is very loose at the base and at the sternal and vertebral borders, to allow for forced lung expansion. The portions of the pleura not occupied by the lung during ordinary respiration are known as complemental sinuses or spaces We find one such space at the base of each lung and also at the quadrilateral space.

The parietal layer or *costal pleura* extends from the roots of the lungs forward, covering the sides of the pericardium to the chest wall in front, and backward to the side of the vertebral column (mediastinal pleura); below, it covers the vault of the diaphragm (diaphragmatic pleura). Normally, the visceral and parietal layers of the pleura are in close apposition to each other, separated only by a small amount of secretion which acts as a lubricant, thus allowing free movement. In disease, the pleural surfaces may be separated by fluid or air, or they may become adherent.

The Trachea and Bronchi

The *trachea* in its downward passage through the middle of the suprasternal region is deflected a little to the right of the median line by the aortic arch *It terminates at its bifurcation into a right* and *left* bronchus at the level of the second ribs (angle of Louis) or fourth dorsal vertebra

The Bronchi: The right bronchus differs perceptibly from the left, which to some extent accounts for the variation in the physical signs obtained from the right and left lungs

Right Bronchus

1. Larger caliber
2. Follows the direction of the lower part of the trachea. Enters the lung opposite the fifth dorsal vertebra
3. Shorter in length (one inch).
4. Lies under the second rib.
5. Gives off its first branch behind the upper border of the third costal cartilage ½ inch from its bifurcation and before the primary has entered the lung tissue
6. Is in relation with the vena azygos, superior vena cava and right pulmonary artery.

Left Bronchus

1. Smaller caliber.
2. Takes a nearly horizontal course and leaves trachea with a sharp change of direction Enters the left lung opposite the sixth dorsal vertebra.
3. Longer than the right.
4. Lies under the second interspace
5. Gives off its first branch twice as far from the bifurcation (one inch) and after it has entered the lung tissue The arch of the aorta encircles the left bronchus at its origin.
6. Crosses the esophagus, thoracic duct, and descending aorta, and is in proximity to the pulmonary artery

Peribronchial lymph glands occur in clusters; they vary in size from that of a millet seed to that of a pea. The large ones lie at the bifurcation of the trachea. In glandular tuberculosis, Hodgkin's disease, and some lung and bronchial affections, these glands may attain a large size. These lymph nodes are situated between the divisions of the bronchi, at the root of the lungs and about the bifurcations of the trachea

Diaphragm

The diaphragm is a powerful respiratory muscle. It is a dome-shaped musculomembranous sheet which separates the thoracic from the abdominal cavity. At its origin it is on a level with the sixth ribs or intercostal spaces anteriorly, and the eleventh ribs posteriorly.

ratory rate and rhythm and the degree of the chest expansion being kept under observation. The movements of one side of the chest should be compared anteriorly, laterally and posteriorly with those of the corresponding part of the other side.



Fig 1—Inspection for symmetrical respiratory chest movements Two pieces of cotton previously dipped in vaseline are placed upon corresponding points of each lateral half of the chest

In order to bring out more clearly any difference between the expansion of one part of the chest as compared with the corresponding part on the other side, a small piece of cotton previously dipped in vaseline or other sticky substance may be placed upon corresponding points of both sides, or the corresponding points may be marked with a colored pencil, thus facilitating the detection of apparently minor delays or restrictions in respiratory expansion.

When the *infraclavicular* regions are to be inspected for uneven expansion, the patient is placed upon a chair or stool facing the light, with the head somewhat lowered. A line is drawn with a colored pencil immediately below the inner two-thirds of each clavicle. The examiner stands directly behind the patient looking downward, choosing a position which will enable him to see both lines simultaneously. The lines should not be visible during expiration, but should come into his range of vision during inspiration. The line that is last visible during inspiration denotes delayed expansion on that side.



Fig 2—Inspection of upper portion of [illegible] they come into view.

Posteriorly, delayed *and diminished* expansion is easily *noted by watching* the play of the *scapulae. It is often* necessary to have the patient breathe deeplv in order to bring out more clearly discrepancies in the respiratory excursion

CHAPTER X

Physical Examination of the Respiratory System by Inspection and Mensuration

Having proceeded with the general and local examination, until the thorax is reached, special *attention is directed* to the examination of the chest, because inspection, palpation, percussion and auscultation are of particular value in the examination of the thoracic organs

Inspection is the act of examining a patient by the sense of sight, comparing the part under examination with one's mental picture of a similar healthy part, and one side of the body with the corresponding part of the opposite side It is quite natural that inspection should be the first method of procedure in a physical examination of the thorax, because the eye will recognize outward conditions long before the other senses can be brought into activity It is, therefore, of great importance in examining the thorax to practice inspection thoroughly and systematically.

Rules to Be Observed During Inspection

1. The patient must be stripped to the waist, otherwise accurate inspection is impossible. If an overmodest female patient refuses to bare her chest in its entirety, one portion at a time may be uncovered and thoroughly inspected

2 The patient must assume a perfectly natural and unconstrained position It is preferable, whenever possible, to have the patient in the *erect posture,* the arms hanging naturally at the sides *Mental and physical ease are important,* and these may often be accomplished by engaging the patient in a general conversation, so as to keep his mind off his own body.

If the standing posture is not possible, the next choice is the *sitting posture* The patient is to sit erect, arms hanging *loosely at the sides, head somewhat elevated,* but muscular rigidity should be carefully avoided. When the lateral surface of the chest *is inspected the patient's* hands should be clasped behind his head, allowing free exposure In a very sick patient the *recumbent posture* is the only possible one, the patient lying entirely relaxed. When lateral and posterior views are required of such a patient he should be gently turned from one side to the other, the facial expression being meanwhile noted for any signs of pain or distress. The effect upon respiration should also be observed during this procedure.

3. The chest is examined anteriorly, laterally and posteriorly with equal care and attention The color of the skin general development, musculature, and the size, shape and symmetry of the thorax are to be noted. First the chest is studied as a whole, then the regions of the one side are compared with the corresponding regions on the opposite side.

4. The whole chest should be exposed to a strong steady light, preferably daylight, *so as to avoid confusing* shadows The surface of the chest under examination should always be turned towards the examiner.

5. During the examination respiration should be uninterrupted, the respi

7. The suprasternal depression is small.

8. The epigastric angle, the space formed by the junction of the coalescing ribs with the sternum, is a right angle.

9. The anteroposterior diameter, sternovertebral, equals about three-fourths of the transverse diameter.



Fig. 5—Normal female chest.

10. The ribs as they leave the sternum are horizontal, but gradually slope upward, being nearly oblique when they reach the spinal articulation

11. The interspaces are wider anteriorly than they are laterally and posteriorly; they are neither prominent nor markedly depressed

12. The spine presents a very slight curvature to the right at the midback; the vertebral spines are not very prominent.

13 The scapulae lie nearly flat upon the ribs when the arms are held in the normal anatomical position.

14 The thorax, excluding the shoulder attachments, is conical in shape, the smaller end being uppermost, gradually increasing in depth as it descends, because of the greater curve and angle of each succeeding rib as it joins the sternum.

Irregularities that may occur in a normal chest are:

1. Prominent clavicles and Louis' angle, thereby causing deep supra- and infraclavicular depressions, are usually seen in individuals who have very thick bones and high cheek bones.

2. Occupational deformities, such as funnel chest (Trichterbrust), a sinking in of the lower portion of the sternum, are often seen in shoemakers and harnessmakers.

3. Shallow upper portion of the chest with a gradual deepening and widening lower portion is often congenital.

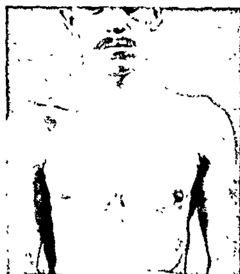


Fig. 6—Irregularities of chest within normal limits Note supra- and infraclavicular depression and deep infrasternal depression

4. Short chest, but with an acute epigastric angle is also often congenital

5 Local irregularities, due to such causes as a badly-united fracture or cicatrices resulting from burns and scalds.

6. Irregularities in the contour of the chest, bulgings, depressions, pulsations, distended vessels, and enlarged glands should be noted. This is best accomplished in the following manner: The examiner should stand about three to six feet in front and away from the



Fig. 3—Inspection of chest and upper abdomen for slight irregularities and pulsations The patient lies supine and the examiner brings his eyes on a level with the patient's body.

patient, with his back to the light, except when slight variations in the upper part of the chest are to be investigated, and then he should stand behind or at one side of the patient, so that he may be able to look downward.

When the patient is in the recumbent position it is often necessary for the examiner to bring his eyes to the level of the patient's chest and upper abdomen, in order to detect more readily slight variations in expansion and feeble pulsations.

The Normal Chest

The ideal chest, such as we are accustomed to attribute to an Apollo or a Venus, is rarely, if ever, encountered in actual practice. If we examine a hundred normal chests, we shall very likely find that no two have the same measurements, yet each one is within the normal limits. The difference in chests is like the difference in facial expressions. A hundred Chinamen will present a hundred different faces, whereby each one can be distinguished from the others, still every face will be of the Chinese type. The same is also true concerning chest and body development

Characteristics of the Normal Chest

1. The chest is usually symmetrical on both sides though slight asymmetry may occur, described under another heading

2 The clavicles are somewhat prominent.

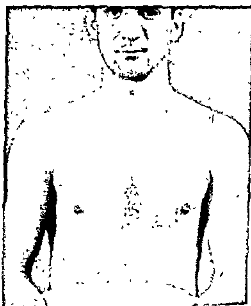


Fig 4—Normal male chest.

3. The supra- and infraclavicular fossae are slightly depressed.

4. Louis' angle is visible (second costosternal junction).

5. The sternum is *nearly straight.*

6 The shoulders are nearly horizontal.

minute depends upon their age; at birth it is about 40 to 50; at the end of the first year, 30; and at the fifth year it is about 26 per minute Respirations are less rapid in the recumbent than in the sitting, and most rapid in the erect posture.

The respiratory rate may become accelerated or retarded as a physiologic or pathologic process Acceleration is more common than retardation. The rate may increase to 30, 40 or even to over 50 per minute; generally, however, it rises no higher than 40 Physiologic increase in frequency of the respiratory rate may be brought about by physical or mental exertion, or by both *Physical exertion*, such as rapid walking, running, mountain climbing, running upstairs, hopping, jumping, "setting-up exercises," heavy lifting, swimming, or any muscular exertion will accelerate the respiratory rate The trained athlete can endure a much greater strain before any change in the respiratory rate is noted than can the man of sedentary habits Convalescents from protracted or grave diseases show a marked increase in the respiratory rate from trivial exertion, such as sitting up in bed The ratio between respiration and heartbeat is usually maintained in these conditions, both being accelerated. *Mental excitement*, such as anger, anticipation of any unusual event, sudden fright, self-consciousness in the presence of strangers, "stage fright," in fact, any condition that will cause a more rapid heart action, will produce rapid respiration

Respiratory Movements

During inspiration the lungs take up approximately 350 to 500 cc of tidal air; this causes each lung vesicle to expand, in consequence of which both lungs balloon out. In order to accommodate them the chest cavity must necessarily become larger. This is accomplished by: (*a*) The descent of the diaphragm, except at its central tendon and (*b*) the raising of the ribs, the upward and forward movement of the sternum, and slight expansion of the intercostal spaces.

The inspiratory act causes the ribs to assume a nearly horizontal plane anteriorly and to some extent laterally, but there is very little change in position posteriorly because the costospinal articulations are fixed and act as a fulcrum to elevate the sternum and its attached ribs Posteriorly, inspiration is noted by the separation and ascent of the scapulae and slight filling of the interspaces. Forced inspiration is accomplished by bringing into play the accessory muscles of respiration, thus lifting the thorax still higher, and causing a greater descent of the diaphragm.

The expiratory act, because of the collapse of the lungs and the ascent of the diaphragm, causes a descent of the ribs, a slight retraction of the intercostal spaces and greater acuteness of the epigastric angle. Posteriorly, expiration is noted by the approach and descent of the scapulae and the lowering of the shoulders.

The *inspiratory movement*, therefore, consists of *expansion* and *elevation* of the chest, and *lowering* of the diaphragm

The *expiratory movement* consists of *retraction* and *recession* of the ribs and interspaces, *elevation* of the diaphragm, and recoil of the lung tissue.

The Diaphragmatic Movement: In repose, the diaphragm is arched upward and assumes the shape of an inverted basin and its sides are in close contact with the inner wall of the thoracic cavity, from its attachment to the level of the fifth intercostal space. During in-

Respiration

Respiration is a process in which atmospheric air is taken into the lungs for the purpose of aerating the blood, and charged or vitiated air is exhaled. The atmospheric air (inspired air) contains approximately 20 to 21 per cent oxygen, 79 per cent nitrogen, and 0 04 per cent carbon dioxide. The expired air contains about 14 per cent oxygen, about 80 per cent nitrogen saturated with water vapor and about 5 6 per cent carbon dioxide. The composition of the expired air varies with the amount of activity, the general metabolic process, and the kind and quantity of food taken in. On an average diet during comparative rest about 900 grams of carbon dioxide are expired daily; during exertion it may exceed 1200 grams.

Tidal air is the amount of air inspired or expired with each respiration during normal quiet breathing; it is about 350 to 500 cc.

Complimentary or complimental air is the volume of air that can be forcibly inspired after a normal inspiration; it is about 1500 cc. or slightly over.

Supplemental or reserve air is the amount of air that can be forcibly expired after normal expiration; it is about 1500 cc. or slightly over.

Residual air is the amount of air constantly remaining in the lungs that cannot be forced out by the deepest possible expiration; it is about 1000 to 1500 cc.

Vital capacity is the greatest volume of air that can be forced out of the lungs after the deepest possible inspiration; it is the sum of the preceding figures and averages in the male 3500 to 5000 cc.; in the female 2500 to 3700 cc.

The blood is brought to the lungs by large arteries (the pulmonary arteries) which divide and subdivide, following the ramifications of the bronchial tree, until at last the smallest capillaries come in contact with the fine air vesicles, the blood and air being separated only by a thin membrane which permits osmosis. After an interchange of gases in the capillaries, the aerated blood in the lungs is finally carried away by increasingly large veins, until it reaches the left ventricle from which it is distributed throughout the body. The blood is brought to the lungs at a definite velocity, depending upon the rate of the heart, usually about 72 heartbeats per minute The air is also brought to the lungs at a definite rate of speed, about 18 respirations per minute, taking in approximately 30 cubic inches or from 350 to 500 cc of air during an ordinary inspiration excursion. The ratio between the respiration and the pulse beat is one to four In other words, the air drawn in by one act of respiration takes care of the quantity of blood brought to the lungs by four heartbeats The respiratory rate and rhythm may be to a large extent controlled by the will. It may be voluntarily deepened or made superficial, accelerated, retarded, or even arrested for half a minute, a minute, or even longer. Therefore, the patient should not be made acquainted with the fact that the examiner is counting the respiratory rate

Normal Respiratory Rate

The respiratory act consists of an inspiratory movement and a short pause, followed by an expiratory movement. These movements occur regularly and rhythmically and are symmetrical on both sides of the chest In the *male* they occur 18 to 20 times a minute. In the *female* 20 to 22 times a minute. In *children,* the number of respirations per

Types of Normal Respiration

The preponderance of upper or lower chest expansion during inspiration and its accompanying contraction during expiration mark two distinct types of respiration observed normally in the two sexes: (I) *Superior thoracic* or *costal* breathing in women; (II) *costoabdominal* or *inferior thoracic* breathing in men

I. **Superior Thoracic or Costal Type of Breathing in Women:** The expansion of the thorax occurs largely in the upper part, and is chiefly produced by the action of the intercostal and scaleni muscles; as the diaphragmatic contractions are slight, they produce only a feeble expansion of the lower portion of the thorax and upper abdomen. Trained singers and orators, by diligent practice, bring the diaphragm into forcible play, thus increasing their lung capacity and causing their breathing to assume a nearly costoabdominal type, at the same time also retaining the supracostal type. The supracostal type of breathing in women was formerly attributed to tight lacing, but this is probably not true, because though the tight lacing has ceased to be fashionable this type of respiration is still present in civilized women and in women of the primitive races who do not, and probably never did, constrict their waists. It is no doubt due to the action of the intercostals and scaleni muscles and the greater flexibility of the female ribs which may be nature's method of allowing sufficient room in the abdomen for childbearing.

II. **Costoabdominal or Inferior Thoracic Type:** In men, the diaphragm is the most important muscle of respiration; when relaxed it projects upwards like a dome into the thoracic cavity, but when contracted during inspiration it becomes flattened and descends, pushing the abdominal viscera before it, elevating the upper part of the abdominal wall and expanding the lower half of the thorax.

These respiratory types are greatly influenced by age, occupation, habits and pathological conditions. In old age when the ribs and cartilages are ossified, respiration is almost entirely abdominal, even in women. In persons following such occupations as singing, wind instrument playing, or glass blowing, both the supra- and infracostal types are found to be well developed Sedentary habits which induce shallow breathing will cause but slight contractions of the diaphragm, even in men.

Respiratory Rhythm

The ratio between the *inspiratory act* and the *expiratory act* is six to seven

The inspiratory act is slightly shorter than the expiratory act. A very short pause follows inspiration; almost as soon as inspiration is completed expiration begins. The pause following expiration is longer than the inspiration pause.

Be it remembered, however, that the normal *inspiratory sound* (the sound heard during normal inspiration) is three times longer than the *expiratory sound*

Mensuration of the Normal Thorax

Mensuration is employed to determine more accurately · (I) The circumference of the chest, and to note its relation to the general build of the individual; (II) the degree of respiratory expansion; (III) the irregularities of the chest and the relative size of either side; (IV) the diameters of the thorax in relation to its circumference.

spiration the diaphragm flattens out and permits the descent of the bases of the lungs in its wake. During expiration, with collapse of the lungs, the diaphragm rises. The deeper the inspiratory act, the lower the descent of the diaphragm and *per contra,* the greater the expiratory act the higher does the diaphragm rise. When the individual assumes a lateral posture, the diaphragmatic excursions are greatest on the dependent side.

Accessory Muscles of Respiration

Normally, the ordinary respiratory muscles—intercostals, diaphragm and, in the female, the scaleni—carry on respiration. Greater depth of respiration is accomplished by increased action of these muscles, assisted by the accessory muscles, thereby producing greater chest expansion. The accessory muscles of respiration are divided into two groups: (I) *Accessory muscles of inspiration,* and (II) *accessory muscles of expiration.*

I. Accessory Muscles of Inspiration:

(*a*) The muscles of the upper respiratory tract, *the levatores alae nasi* and the *levator palati mollis,* enlarge the opening of the upper respiratory tract, thus more readily permitting the passage of air into the larynx. The *sternohyoid, sternothyroid, thyrohyoid* and *omohyoid* muscles depress the larynx, thus facilitating the entrance of air into the lungs The *cricoarytenoidei postici* by their contraction separate the arytenoid cartilages, thereby dilating the *rima glottidis*

(*b*) The respiratory muscles of the neck are the *scaleni* and the *sternomastoids.* The *anterior* and *middle scaleni* raise the first rib, the *posterior scalenus* the second. The *sternomastoids* elevate the sternum and clavicles when the head is fixed.

(*c*) The *pectoralis,* major and minor, when the head and shoulders are fixed, elevate the second to the sixth ribs inclusive. The *serrati postici superiores* elevate the upper ribs. The *subclavius* raises the first rib when the clavicle is stationary. The *levatores costarum brevi* and *longi* draw the posterior portion of each rib toward the spinal column

(*d*) The *levator anguli scapulae,* that part of the trapezius which rises from the occiput and is inserted into the clavicle and acromion, and probably also the *serrati antici majores,* act as inspiratory muscles, inasmuch as they move the lower and middle ribs upward and outward when the shoulder is fixed.

(*e*) The *elevatores* of the head and spinal column aid respiration in cases of croup, spasm of the glottis and when asphyxia is threatening.

II. Accessory Muscles of Expiration: Expiration is usually accomplished *by the collapse of the air vesicles in the* lungs and the upward movement of the diaphragm When the elasticity of the alveoli is lost, muscular action has to be brought into play in order to compress the thorax The principal expiratory muscles are those of the abdomen, which push the abdominal organs upward toward the diaphragm.

(*a*) The *transversalis muscle* shortens the transverse diameter of the abdomen and the *recti muscles* shorten the long diameter.

(*b*) The *internal* and *external obliqui* and *triangularis sterni* depress the anterior part of the lower ribs

(*c*) The *serrati postici inferiores,* antagonists of the serrati postici superiores, depress the four lower ribs, and the *quadratus lumborum,* the lowest rib

Diagrams of Normal and Pathological Chests

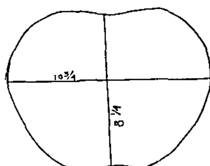


Fig. 8—Normal adult chest

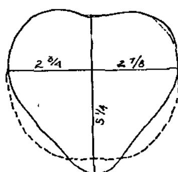


Fig 10—Pigeon chest, child aged 14 months

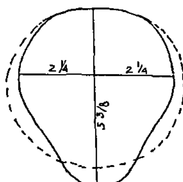


Fig 9—Rickety chest, child aged 15 months

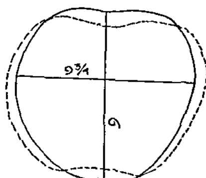


Fig. 11—Emphysematous chest.

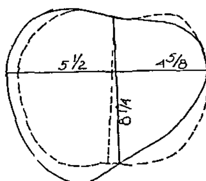


Fig. 12—Fibrosis of the left lung, man aged 30 years

Reduced cyrtometric tracings of the various forms of chest taken at the level of the sternoxiphoid articulation The figures represent measurements in inches, the dotted lines indicate the normal shape of the same chest (After *Sawyer.*)

I. **Circumference of the Chest:** This is obtained by encircling the thorax with an ordinary tape measure or a thoracometer at the level of the third rib anteriorly during quiet breathing. This procedure is known as "thoracometry." The circumference of the thorax at the level of the nipples in front and the lower angle of the scapulae behind when the arms are raised should correspond to half the length of the body. In old age the lower circumference is greater than the upper. The approximate relation between the size of the chest and the height and weight of the individual is given in the following table:

Relation Between Size of Chest and Weight and Height
(After *H Anders*)

HEIGHT	CHEST	WEIGHT
5 feet	33 inches	115 pounds
5 " 1 inch	34 "	120 "
5 " 2 inches	35 "	125 "
5 " 3 "	36 "	130 "
5 " 4 "	36½ "	135 "
5 " 5 "	37 "	140 "
5 " 6 "	37½ "	143 "
5 " 7 "	38½ "	146 "
5 " 8 "	39 "	149 "
5 " 9 "	39½ "	152 "
5 " 10 "	40 "	155 "
5 " 11 "	40½ "	158 "
6 "	41 "	161 "
6 " 1 inch	41½ "	164 "
6 " 2 inches	42 "	167 "
6 " 3 "	42½ "	170 "
6 " 4 "	43 "	173 "

As a general rule it may be remembered that a person measuring five feet has a chest circumference of 33 inches, and weighs 115 pounds, 5 x 3 – 15

For the increase of each inch in height, add one inch to the circumference and five pounds to the weight until five feet four inches. After that add ½ inch to the circumference and three pounds to the weight for each additional inch in height.

The size of the chest circumference does not necessarily indicate the condition of the lungs. Thus, we may have a chest circumference of three or four inches above the normal standard with poorly functionating lungs as in emphysema, and at times a chest circumference of one or two inches below the normal



Fig 7—Technic for measuring the circumference of the chest and chest expansion

standard may shelter perfectly good lungs. The degree of thoracic expansion rather than its circumference is an indication of lung capacity

II. **Degree of Respiratory Expansion:** This is obtained by encircling the chest with a tape measure at the level of the third rib The patient is instructed to take a very deep breath, during which time the measurement is read; he is then instructed to exhale, the tape being drawn in as the chest sinks, and the reading is taken at the end of the expiration. The difference between forced inspiration and forced expiration represents the degree of ex-

of the chest In women the measurements are approximately 23 to 24 cm (9 05 to 9.44 inches) in the upper and lower parts of the chest, and about 1 cm. (0.39 inches) additional when measured a little above the mammae.



Fig. 14—Technic for measuring the anteroposterior diameter of chest.

(*c*) *Anteroposterior diameter* (the depth) is represented by a line passing from any point on the anterior surface to a corresponding point posteriorly. This is usually taken from the sternum to the spinal column, and is, therefore, often called the *sternovertebral diameter* This diameter usually measures 16 cm. (6 29 inches) superiorly and 19 cm (7 48 inches) in the middle and inferiorly. In the aged the inferior diameter is often greater than the superior, due to the flaring out of the ribs. At times other measurements are taken in order to compare one portion of the chest with the corresponding portion on the opposite side; such measurements may be the depth at the apex, from the clavicle to the spine of the scapula; the distance between the sternum and the nipples or between the nipples and the vertebral column, etc.

Pathologic Thorax

Having by inspection become acquainted with the (I) size, (II) shape, (III) symmetry and (IV) respiratory movements of the normal thorax, we are now in a position to appreciate its pathological variations Abnormalities of the thorax in size, shape, or symmetry may be either congenital or acquired.

I. Size

The chest may be abnormally increased or diminished in size

If increased, all diameters are larger than normal, the lungs are overstretched



Fig 15—Posterolateral view of emphysematous chest

emphysematous, and an individual presenting this anomaly is spoken of as being "deep chested" Glass blowing, playing wind instruments, or other occupations requiring pulmonary strain

pansion. In men the average expansion is about three inches; in women two and a half inches. Training generally increases the chest expansion. It is not at all unusual to see athletes who have four to five inches of chest expansion. Expansion less than two inches may be considered pathological, unless there are obvious factors to account for it.



Fig. 13—Technic for measuring transverse diameter of chest

III. Irregularities of the Chest and the Relative Size of Either Side: This may be obtained by the use of the thoracometer, the cyrtometer or the pantograph. The practiced eye will usually detect asymmetries and irregularities without instrumental aid. The instruments of precision are employed for the sake of accuracy.

The *cyrtometer* is a chain of stiffly moving links; it is first molded around the chest, then carefully removed so as to preserve the general outline of the chest at that level. Another instrument, which is less cumbersome and more practical, consists of two narrow pliable metallic bands joined by hinges to a padded saddle which fits the spine. The bands are carefully drawn around the chest until they assume its outline; the anterior junction of the bands is marked; the pieces are then carefully separated and removed from the chest to a piece of paper where they are again put in position, and a pencil tracing is taken from the inside of the strips. The outline of this level is thus obtained.

The relative size of either side of the chest is easily obtained by the use of an ordinary pelvimeter (thoracometer when employed for chest measurement). With this instrument the diameter of each half of the chest is taken and compared. The anterior, posterior and lateral diameters may also be taken with the pelvimeter, and each diameter marked on paper in its corresponding position; a line uniting these points will show the shape and size of the circumference of the chest at that level.

The *pantograph* is an instrument employed by photographers to enlarge pictures. For chest measurements the ends are reversed, the larger end is traced around the chest, while the smaller end, which is fitted with a pencil, transfers this tracing in smaller form, though accurately, upon a sheet of paper.

IV. The Diameters of the Thorax: (*a*) The *long diameter* is measured from the clavicle to the base of the chest This diameter is variable, so much so that it is hardly possible to standardize its normal length

(*b*) The *transverse diameter* (the breadth) is represented by a line drawn from a given point on one side of the lateral aspect of the chest to a corresponding point on the opposite side. In adult men this usually measures 25 cm (9.84 inches) in the upper part and about 26 cm. (10.23 inches) in the lower part

(*e*) The intercostal spaces are narrower and depressed.

(*f*) Louis' angle is very prominent.

(*g*) The clavicles are prominent.

(*h*) The supra- and infraclavicular fossae are depressed.



Fig 17—Phthisinoid chest.

(*i*) The scapulae stand out wing shaped, therefore the name "alar thorax."

One may have a congenital phthisinoid chest, but with proper care may never contract pulmonary tuberculosis. Such a person is perhaps predisposed to this disease, but may not necessarily contract it However, it is true that the majority of phthisinoid chests are found among the tuberculous This form should not be confounded with the *phthisical chest*, which is the product of advanced pulmonary tuberculosis.

III The Phthisical Chest: This type is acquired. A perfectly normal appearing chest in a person who is suffering from active pulmonary tuberculosis may, in time, come to present the characteristics of the phthisical chest. This deformity is no doubt due to deficient lung expansion, which causes collapse and partial atrophy of the intercostal and other chest muscles

Characteristics

(*a*) The chest is generally emaciated

(*b*) The anteroposterior diameter is shortened.

(*c*) Flattening of the chest above the third rib is in evidence.

(*d*) Supra- and infraclavicular depressions are deep.

IV. The Rachitic Chest: Rachitic deformities of the chest may be caused by violent muscular action upon the improperly developing chest of the rachitic child, and by improper calcification. Although many deformities may exist, three distinct varities are recognized:



Fig 18—Rachitic chest showing rachitic rosary.

1. The simple rachitic.
2. The pigeon breast, or chicken breast.
3. The transversely constricted chest

may eventually cause such enlargements. Mountaineers are usually deep chested.

If diminished, all diameters are symmetrically decreased. This condition is usually congenital, although in some instances the chest may be arrested in its development because of insufficient lung expansion in apparently normal individuals

The thorax usually accommodates itself to the size of the lungs; if the lungs are abnormally large, the thorax is also large; small lungs naturally require a smaller lodging place, consequently a smaller thorax.

II. Shape

The alterations in the shape of the chest may be classified as ten distinct pathological types

I **The Barrel-shaped or Emphysematous Chest:** This type is striking in its appearance, occurring in emphysematous persons and is often seen in *those suffering* for a long period of time from continuous attacks of asthma. The emphysematous chest is most frequently observed in persons of, or beyond, middle life. The sufferer has the appearance of a person walking about during a continuous deep inspiration

Characteristics:

(*a*) The chest is short (due to the elevation of the ribs).

(*b*) The chest is full, the greatest fullness occurring in the scapular regions.

(*c*) The shoulders are elevated and are nearly horizontal, because of the elevation of the ribs

(*d*) The neck is short, because of the elevation of the shoulders.

(*e*) The anteroposterior diameter is as long or longer than the transverse; this is caused by the arching forward of the sternum, and the arching backward of the spine, which give it a barrel-shaped appearance.

(*f*) The ribs are massive and horizontal.

(*g*) The interspaces are wider and somewhat bulging.

(*h*) The epigastric angle is obtuse



Fig 16—Emphysematous chest, shoulders high, no supra- or infraclavicular depressions.

(*i*) The scapulae lie flat upon the ribs and are thrown upward, outward and forward.

II. **The Phthisinoid, Alar, Pterygoid or Paralytic Chest:** This type is just the opposite of the emphysematous type; it is, as a rule, congenital The phthisinoid-chested person gives one the impression of being constantly in the act of deep expiration.

Characteristics·

(*a*) The chest is long.

(*b*) It is flat or shallow.

(*c*) The anteroposterior diameter is greatly diminished.

(*d*) The ribs are thin and oblique, causing an *acute epigastric angle*, downward *sloping of the shoulders and a long neck.*

diameter and the absence of the normal forward curve of the ribs. The length of the thorax is not abnormally increased. This type is often seen in pulmonary tuberculosis.

VI. **The Scaphoid or Boat-shaped Chest:** This variety of chest is at times



Fig 21—Broad, flat chest; phthisical. Large transverse diameter.

found in patients suffering from syringomyelia, also in the rachitic and as a result of injury. It is characterized by a median depression of the upper anterior chest wall extending from the top of the sternum to about the fifth or sixth rib This hollow is formed by the depression of the sternum and its adjoining costal cartilages

VII. **Spindle-shaped Chest or Fusiform Thorax:** This deformity may be *acquired by tight lacing* It consists of a lengthened or constricted chest which has assumed a spindle shape. The upper part of the thorax is broadened; the waistline is lower and is decreased in circumference; the spinal muscles become atrophied. The thoracic viscera are pushed up higher in the chest, while the abdominal viscera are crowded downward. Since tight laced corsets have "gone out of fashion," the clinical incidence of this chest abnormality has greatly decreased.

VIII. **Chest of Progressive Muscular Atrophy:** This type is characterized by its peculiar box-shaped appearance, the walls being nearly perpendicular. The lower ribs are extremely oblique, and the intercostal muscles are atrophied. The waist is very slender and constricted (wasp waist).

IX. **Gutter Chest:** This type is characterized by a narrow, shallow, vertical groove corresponding to the midsternal line It is due to a forward convexity of the costal cartilages, which causes them to approximate, thus pushing the sternum backward so that it forms this longitudinal furrow. The gutter chest is usually congenital, although it may be acquired after birth.

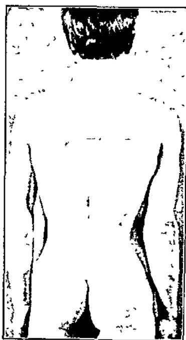


Fig 22—Spindle-shaped chest; rachitic

1. ***The Simple Rachitic Chest:*** This is recognized by the following characteristics:

(*a*) It is shorter and deeper than normal.

(*b*) A shallow depression or groove occurs on either side of the chest and



Fig 19—Rachitic chest, pigeon breast deformity

runs nearly parallel to the anterior axillary line; they correspond to the costochondral junctions; the anterior aspect of the chest is pushed forward, causing the chest as a whole to assume a nearly quadrilateral shape instead of the circular form normal to children.

(*c*) Rachitic rosary is caused by beading of the sternocostal junction This is due to an excessive deposit of limesalts at each sternocostal articulation, causing enlargement of the osteocartilaginous junctional tissues.

2. ***The Pigeon Breast:*** As its name implies, the shape of this type of chest resembles that of the breast of a fowl It is also often compared to the outline of a ship's keel.

Characteristics:

(*a*) The transverse outline is triangular.

(*b*) The sides of the chest are flattened.

(*c*) The lower portion of the sternum is arched forward.

(*d*) The ribs slope sharply backward from their sternal articulation, the angle being straightened at the costochondral junction.

3 ***The Transversely-constricted Chest or Harrison's Sulcus:*** In this type a transverse constriction of the anterior lower portion of the chest below



Fig. 20—Broad, flat, thin chest; small anteroposterior diameter.

the sternoxiphoid articulation is noted, the constriction corresponding to the points of attachment of the diaphragm

V. ***Flat Chest:*** This is characterized by the excessive broadness of the chest, the very small anteroposterior

(*d*) The slight depressions of the intercostal spaces are either obliterated or the intercostal spaces are bulging.

(*e*) The mammary gland is pushed outward, away from the median line.

(*f*) The scapula is also pushed away from the median line.



Fig 25—Emphysematous chest with right-sided pleural effusion

(*g*) The chest movements may be increased, diminished or absent, depending upon the underlying cause of the enlargement

(*h*) The spinal column is bent with its convexity towards the affected side

It should be borne in mind that the spine is always bent with its convexity toward the larger side, no matter whether this be the healthy or the diseased side.

Unilateral enlargement of the chest may be caused by: (1) A foreign substance occupying the thoracic cavity on the affected side; (2) compensatory or vicarious emphysema due to disease of the opposite side; (3) lobar pneumonia; (4) unilateral edema of the skin; (5) subcutaneous emphysema; (6) congenital malformation of the thorax.

1. ***Foreign Substances Occupying the Thoracic Cavity on the Affected Side:*** A large pleural effusion will usually cause elevation of the ribs, flattening out of the intercostal spaces and, in young individuals, the intercostal spaces may bulge somewhat; respiratory motion is limited and at times, entirely absent. The effusion may consist of:

(*a*) *Serous Fluid* (*hydrothorax*): This is a condition caused by certain forms of malignancy of the lung or pleura, by tuberculosis, pneumonia, heart disease after failure of compensation, by acute serofibrinous pleurisy, by nephrosis, and by severe anemia.



Fig 26—Unilateral enlargement due to pleural effusion (Left-sided. The heart pushed to the right as indicated by the cross.) (SEE: p 377)

(*b*) *Bloody Fluid* (*hemothorax*): This is a condition often due to the

X. **Funnel Chest:** In this variety a deep depression is often noted at the lower end of the sternum. It is conical in shape, the larger diameter being in front; the apex is deeply situated and corresponds to the sternoxiphoid articulation. This condition is usually hereditary, though it may occur as a result of rickets.



Fig 23 — Gutter chest, showing deep central groove from third to seventh ribs due to depressed sternum

A lesser and more shallow depression may be of occupational origin, occuring in shoemakers, carpenters, or harness-makers. The constant pressure of a hard object against the lower portion of the sternum, usually continuous from early youth, is responsible for this occupational deformity

III. Symmetry

Normally, both sides of the chest should be symmetrical or nearly so. Pathologically one side may be larger, smaller or distorted. This may be caused either by disease of the underlying viscera, or by disease and congenital deformity of the spine and ribs. Local irregularities of a portion of one side, such as bulgings or depressions may also exist. Whenever an asymmetrical chest is inspected and one side is found to be larger than the other, the question naturally arises which of the two is the normal side. It is therefore necessary to determine whether the apparently larger side is of normal proportions, and the smaller side abnormally contracted; or whether the larger side is hypertrophied and the smaller one of normal proportions

Unilateral Enlargement: The affected side has all the characteristics of an emphysematous chest, *i e.:*



Fig 24—Funnel breast

(*a*) General fullness and bulging on *that side.*

(*b*) Elevation of the shoulder higher than on the normal side.

(*c*) Ribs more horizontal than on the normal side.

stances, however, a tumor in this location *will be accompanied by effusion.* An *aortic aneurysm* may become large enough to cause very decided unilateral thoracic enlargement.

(*h*) *Pericardial Effusion:* Particularly in children, this may cause left-sided chest enlargement



Fig 29—Unilateral retraction due to disease of the chest wall

2. ***Compensatory or Vicarious Emphysema:*** This condition usually arises in one lung as a result of disease in the opposite lung, such as pulmonary atelectasis, fibroid phthisis, fibrinous pleurisy, tumors of the lung, or pleural effusion

The unilateral enlargement caused by compensatory emphysema is often more apparent than real, compensatory emphysema *of one-half of the chest is* usually caused by a retraction of the opposite half; if the diseased side is contracted, the healthy side doing compensatory work enlarges only slightly, but the difference between the two sides is so great that even a moderate increase *in the size* of the sound side makes it appear large in comparison with the contracted side. This, however, is not true of all such cases, because compensatory emphysema of one side as a result of pleural effusion on the opposite side, may produce a bilateral enlargement The two sides may be differentiated by noting the respiratory movements. Compensatory emphysema gives rise to greater chest movement, while in pleural effusion such movement is conspicuous by its absence The results obtained by palpation, percussion and auscultation greatly assist in differentiating pleural effusion from compensatory emphysema.



Fig 30—Unilateral retraction due to *rib resection*

3. ***Lobar Pneumonia:*** Affecting the entire lung this may also cause unilateral enlargement of the affected side, because the lung is the seat of a croupous inflammation The pleura, being somewhat inflamed, causes rigidity of the

presence of a malignant growth in the lung or pleura; or to pulmonary tuberculosis when a small vessel ruptures and stains an already-existing serous effusion; it may also be a result of active inflammation of the lung (as in pneumonia) or of the bronchial glands; of



Fig 27—Pericardial effusion
(SEE p. 470)

stab wounds or other injuries to the chest wall; of the rupture of a blood-vessel or of an aneurysm.

(*c*) *Pus* (*pyothorax*): This condition may be the result of infection of a serous effusion with pyogenic bacteria; it may be a sequel to pneumonia, or to an infectious process such as pulmonary tuberculosis or gangrene of the lung.

(*d*) *Lymph* (*chylothorax*): This may occur as a result of pressure upon or rupture of the thoracic duct.

(*e*) *Air in the Pleural Sac* (*pneumothorax*): This may occur as a result of rupture of air vesicles in the lungs, perforation of a pulmonary cavity, erosion of a bronchial tube or esophagus because of disease, or the introduction of a foreign body causing rupture of the lung. Stab wounds, or other chest wounds, may cause a pneumothorax *either by admitting outside air into the* pleural cavity or by rupturing the lung structure and thus permitting the escape of air. Pneumothorax is often induced as a therapeutic measure (artificial pneumothorax) in tuberculosis and other conditions that may be benefited by putting the lung at rest.

(*f*) *Serous Fluid and Air in the Pleural Cavity* (*hydropneumothorax*), *Pus and Air in the Pleural Cavity* (*pyopneumothorax*): The combination of air and fluid is frequently found in cases of pulmonary tuberculosis, puncture of a lung abscess, pulmonary gangrene, or as a result of stab wounds which have penetrated the pleura.

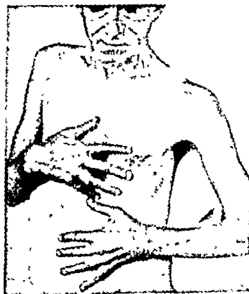


Fig 28—Unilateral retraction of chest due to paralysis of chest muscles

(*g*) *A Solid Tumor:* This may be malignant or benign and may at times attain sufficient size to cause a unilateral thoracic enlargement; in most in-

(*f*) The spine is bent with its convexity towards the opposite (larger) side.

Unilateral diminution in size, if not congenital, may be caused by: (1) Disease of the chest wall; (2) disease of the pleura; (3) disease of the lung; (4) disease of the spine.



Fig 33—Lordosis

1. ***Disease of the Chest Wall:*** This may be due to paralysis of the muscles of respiration, causing atrophy of that side. Primary arrest of development, *e g.*, infantile hemiplegia causes one side to be smaller but its symmetry is maintained

2 ***Disease of the Pleura:*** Chronic adhesions of the pleura prevent proper lung expansions; or in cases of long-continued pleural effusions where absorption is slow, atrophy of the respiratory muscles and fibrosis of the lung may cause retraction because of disuse

3 ***Disease of the Lungs:*** Pulmonary atelectasis, chronic interstitial pneumonia, plugging of a bronchus, or retraction of the lung from any cause may produce unilateral retraction.

4. ***Disease of the Spine:*** The spinal column may be arched forward, backward or bent to either side. This condition may be caused either by disease of the vertebral structures or by their faulty development The arching of the spine produces a general distortion of the thorax in the same direction. Such distortions are classified as follows:

(*a*) *Kyphosis,* bending backwards of the spine (hunchback).

(*b*) *Scoliosis,* lateral spinal curvature The spine may be bent towards either one side or the other, causing a distinct deformity of the ribs.



Fig 34—Kyphoscoliosis with lordosis

(*c*) *Lordosis,* a forward bending of the spine with anterior chondral deformity.

(*d*) *Kyphoscoliosis,* a combination of lateral and posterior spinal curvature (spinal curve).

intercostal muscles, which in turn flattens out the intercostal spaces and slightly raises the ribs The rigidity of the intercostal muscles in pneumonia is analogous to the rigidity of the right rectus abdominis muscle in appendicitis

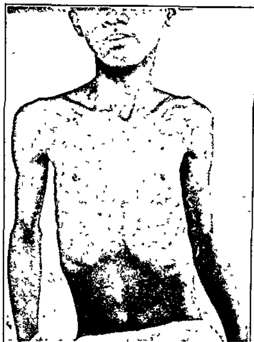


Fig. 31—Left-sided unilateral retraction due to pulmonary atelectasis.

—nature's method of protecting the inflamed viscera from external injury

4. ***Unilateral Edema of the Chest Wall:*** This is often noted in patients suffering from anasarca who persistently lie on one side. The dependent side becomes much more edematous and often presents the appearance of a unilaterally enlarged thorax. The diagnosis of this condition is easily made as the skin pits on pressure

5 ***Subcutaneous Emphysema:*** This may occur on any portion of the body or it may occupy a vertical half of the thorax The author has seen at the Philadelphia General Hospital several such cases which had the appearance of a unilaterally enlarged thorax. On palpation a peculiar crackling is elicited, the skin over the chest is distended to such an extent that the ribs cannot be *differentiated from the intercostal spaces.* There is, however, no interference with respiratory expansion.

Unilateral Diminution in Size or Unilateral Retraction: This condition causes the affected side to be smaller in all dimensions, and to resemble a *unilateral phthisinoid chest.*

(*a*) The chest is drawn in and flattened on the affected side

(*b*) The intercostal spaces are narrowed, depressed and—in extreme cases—the ribs may overlap one another.



Fig 32—Left-sided unilateral retraction, posterior view pulmonary atelectasis

(*c*) The shoulder droops.

(*d*) The mammary gland is drawn towards the sternum

(*e*) The scapula is drawn towards the spine, and stands out wing-shaped

atmospheric pressure the wall sinks. A large superficial cavity of the lung, pulmonary atelectasis, or pleural adhesions may also cause local depression for the same reason.

(*e*) Rachitic deformities

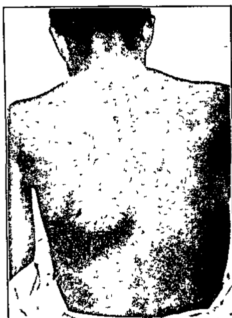


Fig 37—Aneurysm of thoracic aorta

IV. Respiratory Movements

The *respiratory movements* may be pathologically altered in: (I) Type; (II) Amount of chest expansion, (III) Rate, and (IV) Rhythm

I **Type:** The two types of normal respiration, namely, supracostal in women, and infracostal—a mixed costo-abdominal—in men, have already been described (SEE p 233).

Exaggerated bilateral superior costal breathing in women, if not due to emotion or excitement, may be caused by unusual enlargement of the pregnant uterus or by large ovarian tumors, as well as by the same conditions which cause superior costal breathing in men

Superior costal breathing in men may be due to inflammatory conditions of the diaphragm, or paralysis of the diaphragm *preventing its descent during* inspiration. Other causes are ascites, enlarged liver or spleen, or an overloaded stomach which mechanically obstructs the descent of the diaphragm; acute peritonitis, producing rigidity of the abdominal muscles, which in turn prevents lower costal expansion; bilateral pleural effusion and large pericardial effusion. It will be noted that the superior costal breathing in men or abnormal exaggeration in women is due to greater activity of the upper lobes of the lungs, and can be summed up as follows:



Fig. 38—Aneurysm of the thoracic aorta

1. Improper descent of the diaphragm from *any cause, thus throwing the* greatest amount of work upon the upper lobes of the lungs and upon the upper accessory muscles of respiration.

The superficial lines and landmarks are practically valueless in a distorted chest caused by spinal deformities, because the viscera do not retain their normal relations to the chest wall.

Local Irregularities: Local irregularities may consist of bulgings or depressions in any portion of the chest.



Fig. 35—Multiple myeloma.

Local bulgings are readily recognized by inspection; they may be caused by

(*a*) Tumors and cysts of the soft tissues covering the chest wall, by a bony prominence due to a badly united fracture, or by some bone disease or tumor of a bony or cartilaginous structure.

(*b*) Aneurysm of the aorta or other large vessel.

(*c*) Empyema which has burrowed its way to the surface.

(*d*) Mediastinal tumors or greatly enlarged mediastinal glands, causing bulging or necrosis of a bone.

(*e*) Local infections (abscess or "boil" on the chest wall).

(*f*) Hernia of the chest wall with protrusion of a portion of some viscus (lung).

(*g*) Localized emphysema.

(*h*) Upward extension of a subdiaphragmatic abscess, burrowing its way to the surface of the chest.

(*i*) Greatly enlarged liver or spleen.

(*j*) Rachitic deformities.

(*k*) Pleural effusions (in children).

(*l*) Hypertrophied heart (*particularly* in children and young people).

Local depressions may be caused by:

(*a*) Wasting of a muscle from any cause.

(*b*) A broken bone.

(*c*) A very prominent clavicle, giving rise to deeping of the supra- and infraclavicular fossae.

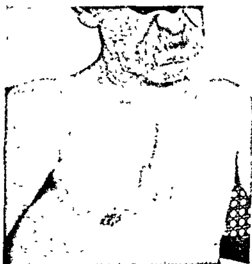


Fig 36—Aneurysm of aortic arch (See · p 531)

(*d*) Localized pulmonary tuberculosis may cause a depression of the overlying portion of the chest wall; the affected portion of the lung is unable to expande and retract that part of the chest wall, and because of disuse and external

condition that interferes with either the entrance of air into the lungs or its exit, will of necessity cause diminished expansion.

(*d*) *Chronic emphysema* In this condition the lung vesicles are overstretched and the vesicular walls have lost their elasticity. The chest is larger than the normal, but respiratory expansion is almost *nil*, because the patient walks about with as much expansion as he can possibly have. He is practically in the act of constantly harboring a deep breath Inspiration brings only the accessory muscles of respiration into play, producing an up-and-down movement of the chest instead of expansion Expiration cannot force the normal quantity of air from the lungs because of the inelasticity of the vesicular walls

(*e*) *Chronic fibroid phthisis*. In this condition the air vesicles are depressed and often filled with fibrous tissue, which encroaches upon the aerating surface and reduces the air space within the lungs, thereby causing diminished expansion. Partial obstruction or spasmodic contraction of the trachea will cause diminished expansion, because it interferes with the entrance and exit of air to and from the lungs

B ***Unilateral:*** 1 *Unilateral increase of chest expansion* is caused by compensatory emphysema due to disease of the opposite lung.

2 *Unilateral diminution of chest expansion* may be caused by:

(*a*) *Pathological conditions of the chest wall* which include pleurodynia, painful condition of the chest wall caused either by a broken rib or an inflammatory focus in the soft structures, or, reflexly, from other parts of the chest wall, the abdomen, or the spinal nerves.

(*b*) *Pathological conditions of the pleura* which may include a thickened pleura, small pleural effusions, localized empyema, or chronic adhesive pleurisy.

(*c*) *Pathological conditions of the lung substance* such as a small consolidation caused by bronchopneumonia, early tuberculosis, specific disease, malignancy, neoplasms in the lung (*i. e.*, tumors, cyst, aneurysm), pulmonary infarcts and small atelectatic areas.

(*d*) *Pathologic conditions of the bronchi,* such as a foreign body, constriction, tumor or a plug of mucus

(*e*) *Combination of any two or three pathological conditions operative in the same case,* such as an injury due to a broken rib or contusion of the soft parts, or the simultaneous occurrence of developmental peculiarities.

3 *Absence of unilateral expansion* may be caused by a large pleural effusion either of blood or pus, or by pneumothorax, massive consolidations, or the plugging of a bronchus with subsequent collapse of the lung, also by pulmonary atelectasis and compression or retraction of the lung.

C. ***Local:*** 1. *Local increase of respiratory expansion* is caused by local compensatory emphysema, *i. e*, a portion of lung is assuming the work of an adjacent part which has been "put out of commission." This condition may occur in a part of the lung adjacent to a consolidation, above a pleural effusion, near an atelectatic area, near a lung compressed by a new growth *Circumscribed expansion* may be due to a large superficial cavity. *Expansion of the intercostal spaces* during expiration is often seen in old cases of severe emphysema or during an asthmatic attack. A lung hernia may at times cause protrusion during deep inspiration.

2. Compression of the lower lobes, thus forcing the upper lobes to do compensatory work.

3. Acute peritonitis preventing upper abdominal expansion so that all the respiratory work must be carried on by the upper lobes alone.

Restricted bilateral chest expansion, or increased costoabdominal respiration is caused by some pathological condition in the upper lobes of the lungs, preventing their proper expansion; consequently, the work of respiration must then be carried on by the lower lobes Among the causes responsible for this condition may be mentioned:

(*a*) Acute pleurisy in the upper thoracic cavity.

(*b*) Broken ribs (upper four).

(*c*) Intercostal neuralgia, herpes zoster and radiculitis producing involuntary rigidity of the chest, thus causing greater abdominal movement.

(*d*) Pericardial effusion

(*e*) Upper mediastinal tumors

(*f*) Aneurysm of the aorta (if very large).

(*g*) Pleural adhesive bands, compressing the upper lobes, and finally,

(*h*) Disease (consolidation or cavity) of both upper lobes

Increased abdominal respiration in infants may be caused by pleurisy or lobar pneumonia, or by Potts' disease (caries of the vertebrae).

Diminished abdominal respiratory movements or increased costal movements may be caused by acute peritonitis or by colic

II. **Chest Expansion:** *Normally* both sides of the chest expand equally on inspiration, though the right side has a somewhat greater expansion than the left. *Pathologically* the following changes may occur:

A. Bilateral { Increase / Diminution } of expansion

B Unilateral { Absence / Increase / Diminution } of expansion

C Local { Increase / Diminution } of expansion

D Wavy expansion.

E Inspiratory retraction

A. ***Bilateral:*** 1. *Bilateral increase of chest expansion* during inspiration occurs only as a result of compensatory emphysema. The upper part of the chest may compensate for the lower or *vice versa*. Increased respiratory expansion of the whole thorax is usually a sign of health rather than disease, because any disease of the respiratory organs will cause a "diminished amount" of expansion.

2. *Bilateral diminution of chest expansion* during inspiration may be due to:

(*a*) *Disease of the chest wall,* such as *paralysis of the chest muscles, or* excessive ossification of the thoracic joints, preventing proper play of the ribs and sternum Intercostal neuralgia, paroxysmal pain in the intercostal muscles, pleurodynia and painful wounds on the surface of the chest will cause voluntary suppression of expansion

(*b*) *Disease of the pleura and diaphragm,* generalized pleural thickening and pleural adhesions, inflammatory conditions or paralysis of the diaphragm.

(*c*) *Disease of the lungs and bronchi,* pulmonary tuberculosis (advanced), fibroid phthisis, pneumonoconiosis, diffuse carcinomatosis and thickened pleura, foreign body in the bronchi, or laryngeal obstruction. Since chest expansion is caused by the rapid interchange of a normal amount of air in the lung, any

condition that interferes with either the entrance of air into the lungs or its exit, will of necessity cause diminished expansion.

(*d*) *Chronic emphysema.* In this condition the lung vesicles are overstretched and the vesicular walls have lost their elasticity. The chest is larger than the normal, but respiratory expansion is almost *nil*, because the patient walks about with as much expansion as he can possibly have. He is practically in the act of constantly harboring a deep breath. Inspiration brings only the accessory muscles of respiration into play, producing an up-and-down movement of the chest instead of expansion. Expiration cannot force the normal quantity of air from the lungs because of the inelasticity of the vesicular walls.

(*e*) *Chronic fibroid phthisis.* In this condition the air vesicles are depressed and often filled with fibrous tissue, which encroaches upon the aerating surface and reduces the air space within the lungs, thereby causing diminished expansion. Partial obstruction or spasmodic contraction of the trachea will cause diminished expansion, because it interferes with the entrance and exit of air to and from the lungs.

B ***Unilateral:*** 1. *Unilateral increase of chest expansion* is caused by compensatory emphysema due to disease of the opposite lung

2. *Unilateral diminution of chest expansion* may be caused by

(*a*) *Pathological conditions of the chest wall* which include pleurodynia, painful condition of the chest wall caused either by a broken rib or an inflammatory focus in the soft structures, or, reflexly, from other parts of the chest wall, the abdomen, or the spinal nerves

(*b*) *Pathological conditions of the pleura* which may include a thickened pleura, small pleural effusions, localized empyema, or chronic adhesive pleurisy.

(*c*) *Pathological conditions of the lung substance* such as a small consolidation caused by bronchopneumonia, early tuberculosis, specific disease, malignancy, neoplasms in the lung (*i. e.*, tumors, cyst, aneurysm), pulmonary infarcts and small atelectatic areas.

(*d*) *Pathologic conditions of the bronchi*, such as a foreign body, constriction, tumor or a plug of mucus.

(*e*) *Combination of any two or three pathological conditions operative in the same case*, such as an injury due to a broken rib or contusion of the soft parts, or the simultaneous occurrence of developmental peculiarities.

3 *Absence of unilateral expansion* may be caused by a large pleural effusion either of blood or pus, or by pneumothorax, massive consolidations, or the plugging of a bronchus with subsequent collapse of the lung; also by pulmonary atelectasis and compression or retraction of the lung.

C ***Local:*** 1 *Local increase of respiratory expansion* is caused by local compensatory emphysema, *i e.*, a portion of lung is assuming the work of an adjacent part which has been "put out of commission." This condition may occur in a part of the lung adjacent to a consolidation, above a pleural effusion, near an atelectatic area, near a lung compressed by a new growth *Circumscribed expansion* may be due to a large superficial cavity. *Expansion of the intercostal spaces* during expiration is often seen in old cases of severe emphysema or during an asthmatic attack A lung hernia may at times cause protrusion during deep inspiration

2. *Local diminution of respiratory expansion* may be caused by local consolidation, solid tumor, aneurysm, or a large gland compressing a portion of lung, encapsulated liquid effusion, deep-seated cavity in the lung, and localized pulmonary atelectasis. *Diminished expansion at the apices* usually indicates consolidation or fibrosis of the lung apices. *Delayed expansion at one or both apices* is an early sign of pulmonary tuberculosis.

It is important to note the difference between diminished expansion and delayed expansion:

(*a*) *Diminished expansion:* By this is meant that the portion of the chest wall so affected does not attain the same degree of expansion during inspiration as does the corresponding portion on the opposite side. This is often seen over areas of consolidation of the lung, chronic fibrosis of the lung, tumors in the lung, pleuro-pericardial adhesions; in fact, any condition that displaces the normal air with an airless substance will cause diminished expansion.

(*b*) *Delayed expansion* means that the portion of the chest wall so affected does not expand as rapidly as the corresponding portion of the chest wall on the opposite side, but eventually the affected portion attains the same degree of expansion as does the opposite normal side. This condition is found in mild infiltrations of the lung and slightly thickened pleura; it is usually indicative of incipient manifest pulmonary tuberculosis

Diminished expansion is likely also to be delayed; that is, the affected portion begins its inspiratory expansion somewhat later than the sound portion; it rises less rapidly and does not expand to the same extent as does the healthy portion on the opposite side.

D. ***Wavy Expansion:*** Wavy expansion is at times noted over a limited portion of the thorax during the first and third stages of lobar pneumonia and in the massive bronchopneumonias In these conditions there are patches of compensatory air vesicles adjacent to consolidated areas which cause sections of the thorax to expand irregularly, thus producing a wavy effect.

E. ***Inspiratory Retraction:*** Normally, during the first half of the inspiratory act, retraction of the intercostal spaces is noted in the lower portions of the axillary and infraaxillary regions; in the second half of the inspiratory act the intercostal spaces flatten out and are on the same plane as the ribs. Pathologically, the lower intercostal spaces remain depressed during the entire respiratory act, and, in severe cases, the retraction becomes more marked during forced inspiration This phenomenon occurs as a result of bronchial obstruction, which prevents the lung from becoming fully inflated. The location of the area thus affected often indicates the seat of obstruction.

1. *Inspiratory retraction of the suprasternal notch* indicates laryngeal obstruction, often seen in membranous or diphtheritic croup (laryngeal diphtheria), laryngismus stridulus, the lodgment of a foreign body in the larynx, compression of the larynx by an aortic aneurysm, enlarged gland, retropharyngeal abscess, enlarged thymus gland, or a spasmodic contraction of the larynx due to any cause.

2. *Inspiratory retraction of the infrasternal notch* is often seen in attacks of asthma, orthopnea and also in the above named conditions.

3. *Bilateral inspiratory intercostal retraction* of the entire thorax results from

partial obstruction of the trachea above its bifurcation.

4. *Unilateral inspiratory intercostal retraction* is caused by the partial obstruction of a primary bronchus.

5. *Local inspiratory intercostal retraction* is due to partial obstruction of one of the smaller bronchi The lesion which brings about bronchial obstruction may either be situated within the lumen of the tube, or it may cause compression from without.

6. *Inspiratory bulging* above the clavicles, *and in the second and third inter*costal spaces near the sternum, is noted at times in moderately young individuals suffering from chronic emphysema.

7 *Expiratory bulging* of the intercostal spaces and the supraclavicular regions is frequently seen in cases of emphysema and asthma, because the inflated lung is not readily emptied during costal depression. Large pulmonary cavities with adherent walls will often cause local expiratory bulging, when all intercostal spaces excepting those overlying the cavity collapse, so that the pressure of the ribs against the lung causes the cavity to bulge; this, in turn, produces distention of the overlying intercostal spaces This condition can be brought out more prominently by comparison of the affected area with the normally retracted intercostal spaces. In advanced pulmonary tuberculosis, forced inspiration will often cause expiratory bulging of the upper intercostal spaces

Inspiratory retraction and expiratory expansion of the lower intercostal muscles is sometimes noted in long-standing cases of pleural effusion It indicates weakening and relaxation of the intercostal muscles

Local Pulsations and Enlarged Veins (SEE · p. 396).

Edema: The chest wall often becomes edematous in cases of general anasarca, most noticeably upon the dependent portions of the thorax. Inflammatory areas and portions of the thorax from which the circulation has been cut off often present local edema. Urticaria and angioneurotic edema may affect the thorax in a manner similar to that of any other portion of the body This condition may be differentiated by its evanescence, discolorations and the severe itching which accompanies it

Litten's Diaphragmatic Phenomenon Sign: To elicit this sign the patient is placed supine, his chest bared, his hands clasped above his head and his feet pointing towards a window or any other good illumination, so that the light over his feet strikes obliquely from this single source The examiner stands at one side and a short distance from the patient, with his back to the light When the patient breathes deeply, a vermicular movement of a narrow shadow may be observed in the infraaxillary region, from the seventh to the ninth or tenth intercostal spaces, which descends with inspiration and ascends during expiration This shadow corresponds to the diaphragmatic action; during inspiration the diaphragm in its descent separates itself from the inner surface of the thoracic wall in each successive interspace, thus forming a vacuum. This vacuum is soon filled in by the lower portion of the lung, which travels in the wake of the diaphragm and rapidly obliterates the intercostal depressions. Expiration causes this shadow to move upward, but this movement is not always visible. This phenomenon is always observed in healthy persons who are not too stout, and who can relax themselves so com-

pletely as to take full inspirations when directed to do so.

The absence of this sign on both sides may be caused by bilateral pleural effusion, chronic emphysema, fibroid phthisis, and in fact, any condition that would interfere with bilateral expansion.

Absence of this phenomenon on one side only, may be caused by pleural effusion, consolidation of the lung, and pleural adhesions. Extensive tumor formation below the diaphragm and very large ascitic collections may also be evidenced by the absence of this sign, because these conditions may interfere with the descent of the diaphragm.

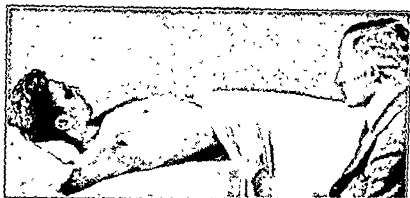


Fig. 39—Watching for diaphragmatic phenomenon (Litten's sign)

This sign is of importance at times in differentiating a right-sided liquid pleural effusion from a subdiaphragmatic abscess, or an enlarged liver. Its absence may indicate pleural effusion.

III. **Respiratory Rate:** The normal respiratory rate in men is 18 to 20 per minute; in women 20 to 22; in the newborn from 40 to 50, and at the fifth year of life about 26 per minute. The respiratory rate may be accelerated or retarded as a result of certain pathological conditions.

Hyperpnea: An increased respiratory rate may occur as a result of: (*a*) Pain; (*b*) febrile disease; (*c*) disease of the respiratory system; (*d*) cardiac disease; (*e*) disease of the abdominal viscera; (*f*) irritation of the respiratory center; (*g*) disease of the diaphragm; (*h*) disease of the blood; (*i*) disease of the kidneys; (*j*) certain constitutional diseases, as acidosis; (*k*) poisoning by certain drugs; (*l*) hysteria and other nervous conditions; (*m*) chest deformities, and (*n*) atmospheric conditions.

(*a*) Pain in any part of the thorax or abdomen which increases during respiration, will cause respiration to be rapid and shallow in order to disturb the affected muscles as little as possible. This is often seen in cases of intercostal neuralgia, broken ribs, painful wounds of chest and upper abdomen, herpes zoster, pleurodynia, pleurisy, myalgia, periostitis and arthritis affecting the thoracic articulation. Acute peritonitis, colic, either hepatic or renal, Dietl's crisis, gastric ulcer, carcinoma of stomach in the later stages, or gumma of the mediastinum or of the sternum, may all cause pain which will increase the respiratory rate

(*b*) Febrile diseases, irrespective of etiology, because of increased oxidation, produce rapid respiration, excepting in the early stages of meningitis and in

certain terminal conditions. In most instances, the respiratory rate does, however, increase in proportion to the severity of the fever. In extreme pyrexia the respiratory rate may equal 30 to 40 per minute; in children 50 to 60, even in the absence of lung complications.

(*c*) *In diseases of the respiratory system*, the respiratory rate is increased out of proportion to the temperature and pulse rate. This is usually due to mechanical obstruction to the interchange of gases in the lungs and to toxins formed in the blood which act upon the respiratory center. The pneumonias and pulmonary tuberculosis are examples of a combination of both conditions. Acute and chronic lung diseases, other than those mentioned; bronchial obstruction by tumor or disease of the bronchial tubes; atelectasis, bronchiectasis, pleural effusions of air, pus or other fluid; plastic pleurisy, mediastinal tumor, *i. e.*, aneurysm, Hodgkin's disease, or enlarged mediastinal gland and emphysema cause increased rapidity of respiration.

In chronic pulmonary diseases where no actual obstruction is present the respiratory rate may not be greatly accelerated Its rapidity often depends upon the nutrition of the patient. Stout persons afflicted with pulmonary tuberculosis usually breathe faster than do emaciated ones who suffer a similar lesion, because the emaciated patient possesses a smaller quantity of blood than does the stout one, so that a smaller quantity of oxygen is required for decarbonization An acute infection superimposed upon a chronic pulmonary disease, *i. e*, emphysema, bronchiectasis, etc., always accelerates the respiratory rate

(*d*) *Cardiac Diseases:* Next to diseases of the respiratory system, disease of the heart is the most prominent cause for rapid respiration, the rapidity of the respiratory rate being directly proportionate to the damage suffered by the heart muscle. Valvular heart disease, cardiac arrhythmia, tachycardia, myocardial degeneration, either fatty, syphilitic or arteriosclerotic, and pericardial effusions usually increase the respiratory rate even when the heart muscle is not badly damaged. This is because any one of these defects forces the heart to greater effort in order to bring the required quantity of blood to the lungs within the normal time for oxygenation; therefore, increased cardiac rapidity usually results in an increased respiratory rate; this is particularly true when an extra effort such as hopping, fast walking, running or when any physical or mental strain is undergone by the patient.

When the heart muscle is weak and can no longer compensate for a defective valve or other abnormal condition, overfilling of the pulmonary circulation or pulmonary stasis takes places, and aeration becomes difficult In order to overcome this stagnation, the lungs attempt to bring as much oxygen in contact with the blood, and to carry away as much carbon dioxide in as short a time as possible, thus causing rapid breathing, and in advanced cases of myocardial weakness, dyspnea, and often, orthopnea, will result. The rapid breathing in such cases is also due to the accumulation of large amounts of carbon dioxide in the blood stream, and this gas has a distinctly stimulating effect upon the respiratory center.

(*e*) *Diseases of the Abdominal Organs:* Ascites, very large liver and spleen; greatly enlarged kidney, due either to tumor (hypernephroma), hydro- or pyonephrosis; ovarian tumor, large pregnancy, distended bowel, tym-

panites, or any condition in the abdomen which causes the diaphragm to be pressed upward into the chest cavity and limiting its movements will cause rapid and shallow breathing.

(*f*) *Irritation of the Respiratory Center:* Tumors of the brain, cerebral hemorrhage and meningitis may at times cause rapid breathing It is then often also irregular as to fullness and frequency.

(*g*) *Disease of the Diaphragm:* Diaphragmatitis, subdiaphragmatic abscess, diaphragmatic hernia and evisceration, partial paralysis of the diaphragm, and, in fact, any condition of the diaphragm that prevents its contraction and relaxation will produce rapid and shallow breathing

(*h*) *Diseases of the Blood:* All forms of anemia, either primary or secondary, will cause rapid breathing; the greater the anemia, everything being equal, the greater the respiratory rate In anemia the oxygen-carrying units of the blood are greatly reduced, thus requiring more frequent visits to the source of oxygen; the consequent accelerated circulation induces an increased respiratory rate.

(*i*) *Diseases of the Kidney:* Acute diseases of the kidney cause increased respiratory rate because of toxins retained in the blood Chronic diseases of the kidney may cause rapid breathing and dyspnea because of the accompanying anemia, retained toxins in the blood, and in some forms of kidney disease because of ascites, pleural effusions and edema In chronic nephritis there may occur at times a retention of acids such as sodium acid phosphate which leads to acidosis and its accompanying hyperpnea.

(*j*) *Constitutional Diseases:* Such constitutional diseases which cause cachexia, anemia, emaciation, pyrexia, or brain disorders, will often produce more rapid breathing. Graves' disease, chronic malaria, diabetes, syphilis, malignant disease, pyemia, etc., are among the constitutional diseases that may eventually cause hyperpnea or dyspnea.

(*k*) *Poisoning by Drugs:* Strychnine, atropine, alcohol, ether, the coal-tar derivatives and most of the respiratory and cardiac stimulants when administered in an overdose will cause hyperpnea.

(*l*) *Functional Nervous Conditions:* Those suffering from hysteria, neurasthenia and other functional nervous conditions are subject to rapid respiration on the least provocation.

(*m*) *Chest Deformities:* Persons with rachitic chest deformity, pigeon breast, scoliosis, kyphosis, lordosis, or Pott's disease have a rapid respiratory rate because of lung compression, the chest cavity not being sufficiently large to permit proper lung expansion.

(*n*) *Atmospheric Conditions:* Close, stuffy rooms, bad air, diminished amount of oxygen in the inspired air, poisonous gases, irritating vapors, or other respiratory irritants cause hyperpnea. High altitudes and caisson work produce an increased respiratory rate, often of such severity as to cause dyspnea.

Dyspnea, Rapid and Difficult Breathing: Dyspnea may be *subjective* and *objective*.

Subjective Dyspnea The person thus suffering is usually of a nervous type and complains of difficulty in "catching his breath" and of a sense of weight and constriction over the precordium or epigastrium. In reality, the respiratory rate is not increased nor is there any difficulty of inspiration and expiration, only occasionally a deep breath is being taken

by the patient. This condition is not true dyspnea; it is a type of air hunger.

Objective or true dyspnea consists of rapid and difficult breathing which may occur both during inspiration and expiration or during either act. The patient is usually somewhat cyanosed, keeping his mouth open; the lips and tongue are dry, and the nostrils dilate with each respiration; the respirations are short, rapid, and difficult, and the accessory muscles have to be brought into action on the least exertion.

This condition may be caused by heart lesions after failure of compensation. It is also seen in severe emphysema, chronic bronchitis, pneumonia, extensive pleural effusion, large abdominal effusions, in enormous hypertrophy of the liver or spleen, or in any condition that seriously interferes with respiration and circulation

Inspiratory dyspnea or difficulty in getting air into the lungs occurs as a result of obstruction of the trachea by a foreign body, a tumor, or an aneurysm of the ascending aortic arch or subclavian artery; spasmodic contraction of the larynx; membranous croup; paralysis of the posterior cricoarytenoid muscle (dilators of the glottis); diseases of the lungs, *i. e*, edema, pneumonia, advanced tuberculosis (particularly in children), sudden collapse of one lung due to pneumothorax, large aneurysm, extensive pericardial effusion, and in extreme cases of kyphoscoliosis

Expiratory dyspnea is characterized by a prolonged labored expiration, followed by difficult inspiration; the face is cyanosed, the eyeballs are bulging, and the abdominal muscles become rigid in their effort to assist in expiration. This condition may occur as a result of a movable tumor situated below the glottis and having a valvular action, the outgoing air pushing it against the rima glottidis, thus causing obstruction, while the incoming air pushes it to one side, thus allowing unobstructed inspiration

Chronic emphysema and bronchial asthma are prominent causes of expiratory dyspnea. The lung vesicles having lost their elasticity cannot recoil properly, and therefore require the aid of the accessory muscles of expiration This condition often results in inspiratory dyspnea; because of the prolonged time required to empty the lungs of their air content, a fresh supply of air is quickly needed, and rapid forcible inspirations result

Orthnopea (Inability to Breathe Except in an Upright Position): The respiratory rate may be rapid or slow. The patient has to brace himself in order to breathe. All the accessory muscles of respiration are forcibly brought into play, the patient being compelled to assume a sitting or standing posture, he is cyanosed, wears an anxious expression and has to struggle for each cubic inch of air he inhales and exhales

This condition is seen in grave cardiac diseases after failure of compensation, bronchial and cardiac asthma, severe cases of emphysema, pneumonia or edema of the lungs Any condition that causes dyspnea, if not remedied, may eventually lead to orthopnea

Paroxysmal dyspnea leading to orthopnea is seen during attacks of angina pectoris, bronchial, cardiac and renal asthma, or spasmodic croup. It may also be caused by a tumor or an aneurysm pressing upon the trachea or bronchus.

Hypopnea (Oligopnea, Bradypnea), Retarded Breathing and Slow Breathing: The respiratory rate may become as slow as six, eight or ten per

minute, and respirations may be very shallow or abnormally deep Hypopnea is usually accompanied by a slow pulse, though in some conditions the pulse rate may be high and the respiratory rate low. Malingering should be excluded before one comes to the conclusion that a patient has hypopnea, because the respiratory rate may to some extent be voluntarily controlled.

Conditions Causing Hypopnea

(a) *Cerebral compression,* such as a depressed fracture of the skull; cerebral, pontine or meningeal hemorrhage; cerebral or cerebellar tumors or abscess; gumma of the meninges; foreign body in the brain due to a gunshot wound or osteomata of the cranium; it also occurs during the early stages of certain forms of meningitis

(b) *Drug Poisoning* Poisoning by opium and its derivatives, by chloral. aconite, antimony, the coal-tar hypnotics, *i e,* veronal, sulfonal, trional, medinal. and acetanilid, by the barbiturates, by chloroform, alcohol, and digitalis is manifested by abnormal retardation of breathing

(c) *Shock and collapse,* whether due to injury, fright, the sudden onset of an acute illness, excessive loss of blood. excessive diarrhea, or surgical operation, fainting or other psychic disturbances, are likely to cause hypopnea

(d) *Constitutional Diseases:* Uremia may at times produce deep and retarded respirations, or very slow and shallow respiration. In some patients suffering from a constitutional disease, the respiratory rate is normal, and in others not infrequently the breathing may be very fast. The difference in the respiratory rate probably depends upon the extent of the toxicity of the blood and its effect upon the respiratory center in the medulla Diabetes mellitus, with impending coma. often produces slow and very deep breathing (air hunger), "Kussmaul's type of breathing."

This peculiar type of respiration which precedes the onset of diabetic coma was first described by Kussmaul in 1874, "and to the clinical picture as he portrayed it," says Foster,[1] "little if anything has been added The respiratory movements are long and deep, involving all the muscles, and suggest in the inspiratory phase the 'long breath that precedes a yawn.' The expiration appears more complete than normal, even forced. With this there may be increase in the respiratory rate, which, however, is usually from sixteen to twenty per minute. The German term 'Grosse Atmung' is exactly descriptive"

"Kussmaul's air hunger," a very similar type of breathing, is observed in states of extreme acidosis, or in the patients suffering from excessive loss of blood, as in postpartum hemorrhage or ruptured ectopic pregnancy

(e) *Functional Nervous Diseases* Hysteria, epilepsy, catalepsy and trance are characterized by partial suspension of animation, with consequent retardation of breathing

(f) *Painful conditions of the chest* often compel the patient to withhold his respiration as long as possible.

(g) *Chronic obstruction of the larynx and trachea* and chronic emphysema (when the patient is at rest) may cause hypopnea.

(h) *Chronic fibrosis* of the lungs (fibroid phthisis) is often a prenatal cause which will produce hypopnea after birth

(i) *Caseous bronchial glands* in children may cause a respiratory rate of

[1] Foster, N. B : Diabetes Mellitus, J. B Lippincott Co, 1915

10 to 12 per minute, the pulse is rapid and the child will usually be found to be undernourished. When the gland is absorbed, or becomes fibrotic, a normal respiratory rate will be established and at times hyperpnea will replace the previously existing hypopnea

(j) During the early stages of certain forms of meningitis the respiratory rate becomes slow.

IV. **Respiratory Rhythm:** *Normally,* inspiration bears a definite relation to *expiration, the two acts being separated* by a pause. The respiratory movements occur regularly and rhythmically.

Pathologically, either inspiration, expiration, or both may be altered in length and duration.

Abnormalities of Rhythm: 1 Sighing (air hunger), a very deep inspiration followed by rapid or broken expiration, may result from habit, particularly in nervous individuals, or from diminished oxidation of the blood, as in partial asphyxia or acidosis It occurs as forerunner of diabetic and uremic coma, and occasionally in gallbladder disease

2 Abnormally shallow and at times irregular breathing is seen in collapse and terminal stages of pulmonary tuberculosis and in acute pulmonary disease.

3. Abnormally deep and irregular respiration is seen in late stages of pulmonary tuberculosis, diabetes, cerebral disease and acidosis.

4 Spasmodic and jerky inspiration and expiration is seen in pleurodynia and pleurisy.

5. *Increase in length of inspiration is* seen in obstruction of the upper air passages

6. Shortened inspiration ending in an expiratory grunt is seen in lobar pneumonia.

7. Increased length of expiration is seen *in asthmatic breathing (and pneumonia).*

8. Lengthened respiratory pause is seen in emphysematous breathing and in oligopnea.

9. Stridulous breathing, *i e,* high pitched, barking, crowing or hissing sounds heard during inspiration may be caused by obstruction of the glottis (internal or external). It also occurs in spasm of the glottis, *i e.,* croup, laryngismus *stridulus* and at the acme of a paroxysm in whooping cough

10. *Cheyne-Stokes breathing* is an arrhythmical type of breathing which follows a fixed cycle, the respiratory movements becoming gradually slower until they finally cease. After a short pause the respiratory movements again commence, at first slowly, gradually increasing in depth and frequency until they become dyspneic. They then gradually become slower and shallower and cease, only to start another cycle In other words, they are paroxysms of dyspnea followed and succeeded by periods of apnea This is seen in cases of central nervous diseases, in coma, and in toxic states; also normally in the aged and in infancy.

11. *Sternomastoid Breathing or "Head-nodding Breathing"* Respirations are irregular and gasping, accompanied by a guttural inspiratory sound. The chin is thrown quickly upward during inspiration and falls slowly during expiration. This type of respiration may be seen in cases where death is imminent.

12. *Meningeal breathing* (Biot's) is an irregular arrhythmical type of breathing resembling the Cheyne-Stokes type, but unlike the latter it follows no definite cycle The periods of apnea and

hyperpnea are irregular in duration and time. Two or three respirations may occur in quick succession, followed by a very long pause. During this pause the patient's muscles relax, the lower jaw drops and the patient appears as if dead. Muscle tone rapidly returns with the next few respirations. This type of breathing is seen in the terminal stage of meningitis, particularly in tuberculous meningitis. In old asthmatic cases with myocardial degeneration, Biot's type of respiration is often observed several hours before death.

CHAPTER XI

Physical Examination of the Respiratory System by Palpation

Palpation is the act of examining an underlying organ by feeling with any part of the hand the overlying surface and is usually the second step in a physical examination It is especially important in the examination of the thorax because it not only confirms or disproves the results of inspection but also reveals certain physical signs that cannot be obtained by any other method.

Technic

In order to be of value in a physical examination palpation must be conducted systematically and with a definite object in mind. In other words, one must know how to palpate and have a definite reason for so doing.

General Rules: 1. The examiner must gain the confidence of the patient and make him or her feel entirely at ease Self-consciousness will invariably cause muscular contractions and rigidity, thereby making palpation worthless. A few friendly words, and not too brusque a manner on the part of the physician will usually suffice to produce the desired relaxation

2. The patient's chest is to be bared of all clothing.

3 The examiner is to assume at all times a perfectly natural and unconstrained position

4 The examiner's hands should be warm and dry; a cold clammy hand applied to a warm body will be sure to produce reflex contraction of the muscles, and greatly mar the results to be obtained by palpation.

5. By the same token, the fingernails should not be long or sharp and the hands should at all times be kept as attractive looking as possible; rings, because they interfere with the tactile sense, should not be worn.

6 The hands should be applied lightly, but firmly, so as to avoid unnecessary tickling of the skin or hurting of the part; pressure may gradually be increased if the case requires it.

7. The patient is to assume an unconstrained position, either standing, sitting or lying. The arms must lie in a natural position and no part of the body under examination should be held rigid, because undue restraint may cause apparent asymmetries.

8 Corresponding parts on the opposite sides of the body should always be compared

Palpating Respiratory Movements

Slight differences of the respiratory expansion between opposite sides may not be appreciated by the eye, but will be detected readily by the trained hand The examiner should lose no opportunity to cultivate as acute a tactile sense as possible, and constantly endeavor to develop it still further.

Palpation should be practiced first during ordinary breathing and later during forced respiration

Anteroposterior Expansion: The palmar surface of one hand is applied anteriorly over the upper part of the thorax, the other hand being applied over the posterior aspect at the same

plane, the fingers separated as far as possible without straining them. The patient standing or sitting with his shoulder pointing towards the front of the examiner is instructed to breathe naturally several times, and then to breathe deeply. The degree of chest ex-



Fig. 1—Palpating for anteroposterior chest expansion.

pansion can thus be judged in this plane.

Lateral Expansion: The examiner places his hands in the patient's axillae while the patient breathes and the gross expansion of both sides is carefully noted. The expansion of the upper axilla should be compared with that of the lower axillary region on the same side. This is to be followed by simultaneously comparing the expansion of the corresponding regions on both sides It should be borne in mind that the expansion in the infraaxillary region on the right side, particularly from the eighth rib downward, is limited, because the liver occupies that position. The same holds true, but to a lesser extent, of the left lower side, which is occupied by the spleen and fundus of the stomach.

Apices of the Lungs: ***Anteriorly*** (supraclavicular fossae): The examiner lightly fits into each supraclavicular space as many of the finger tips of one hand as he can conveniently place there, making use of his hands on either side, according to his position.

When the examiner stands in front of the patient the fingers of his right hand are to palpate the patient's left supraclavicular region and the fingers of the left hand are to palpate the patient's right supraclavicular region. When the examiner stands in back of the patient, the fingers of his right hand are to palpate the patient's right supraclavicular space and the fingers of the



Fig 2—Palpating for lateral chest expansion and tactile fremitus.

left hand are to palpate the patient's left supraclavicular space.

The examiner may stand either in front of his patient or behind him. The latter position is best adapted for handling patients who are much taller than

the physician. To palpate properly in this position the patient should sit upon a convenient chair, slightly supported by the back of the chair, and his arms hanging loosely, or his forearms resting upon his thighs The examiner stands behind him while palpating Care should



Fig 3—Palpating apices of lungs, noting expansion and tactile vocal fremitus.

be taken that no portion of the examiner's hands rests upon any part of the patient's body except the part under examination.

Posteriorly: The part under examination (apex) faces the examiner, the finger tips being lightly placed above the spines of the scapulae

It is of great importance to detect any delayed and diminished expansion, anteriorly or posteriorly, at one or the other apex Such delay or diminution of the expansion may mean a diseased condition of the pleura or the apex of that lung Pulmonary tuberculosis usually first manifests itself at the apex of the lung

Infraclavicular and Mammary Regions: The *examiner stands in front of* the patient; his hands are applied nearly perpendicularly to the ribs, both hands being applied simultaneously, one to either side. When in doubt as to the preponderance of expansion of one side over the other, the examiner's hands *may be crossed, the right hand applied* to the right side of the chest and the left hand to the left side, the examiner facing the patient

Infrascapular Regions: The patient's back confronts the examiner. The hands are applied so that the flexor surfaces of the wrists nearly meet; the fingers pointing horizontally outward, resting in the intercostal spaces Palpation in this region, which is appreciated more by the palm than by the fingers is a valuable adjunct in detecting pleural effusions and consolidation; both conditions *being made conspicuous by the* absence of respiratory expansion, though they can be differentiated by the absence or presence of vocal fremitus



Fig 4—Palpating anterior aspect of chest for chest expansion and tactile fremitus

To determine with a fair degree of accuracy the amount of expansion of

either lower lateral and posterior half of the chest, the following technic should be observed:

The patient stands or sits with his back toward the examiner; the examiner places his right hand on the patient's right side and his left hand on the left side, the hands being so placed that the



Fig 5—Method of noting expansion of bases of lungs

fingers extend well into the infraaxillary regions where they are held firmly. The palmar surface of the hand rests lightly and the thumb is held at right angle to the index finger and adjacent to the patient's spine During inspiration the thumb recedes from the spinal column. The greater the expansion, the further will be the separation of the thumbs. By comparing the distance of each thumb from the spine during inspiration, the difference in the expansion of the two sides will readily be detected. (For the significance of alteration of chest expansion, see p. 250).

Purpose of Palpation

Palpation is employed for a double purpose: *First,* to confirm or disprove certain impressions received by "inspection," and *second,* to elicit physical signs that cannot be appreciated in any other way.

1. Signs Confirming Inspection

The diagnostic importance of bulgings, depressions, skin rashes, scars, pulsations and respiratory movements may be emphasized through palpation.

Bulging: Inspection may reveal that a certain portion of the surface under examination is higher than its surrounding parts; such an elevation is known as a *bulging.* Bulgings may be caused by several conditions: A broken bone improperly set; an exostosis or congenital deformity of a bone; a tumor of the skin or adjacent parts; an aneurysm or a hernia of the lung; also by a greatly hypertrophied heart or a massive pleural effusion.

Technic· In order to determine the character of the bulging so as to arrive at a diagnosis, palpation should be employed in the following manner:

If the bulging is small and appears linear, it should be palpated by feeling the part with thumb, index and middle fingers. The thumb and middle finger are to rest respectively upon the superior and inferior edges of the elevation while the index finger rests upon its center. With the fingers in this position the hand is run several times across the elevation. By this procedure the conformity, consistency and sensitiveness of the part are easily determined. If the bulging is small and circumscribed, one

should begin by feeling it with the index fingers of both hands. The mass is at first gently palpated at its extreme edge with each index finger. If it is found to be hard, it should be grasped firmly, and a gentle attempt made to dislodge it. This is done in order to ascertain its degree of mobility. A mass which is freely movable with its skin, is most likely a superficial tumor or a cyst. If the mass is slightly movable and the skin moves over it, a deep-seated non-inflammatory tumor may be suspected If the mass is immobile and very hard, it is probably a bony tumor. A slightly yielding immobile tumor may be due to a deep-seated aneurysm or malignant growth. A tumor that can be reduced, and reappears after coughing is probably a lung hernia; these tumors are rare, are *self-reducible, and usually occur in* the upper part of the chest, close to the sternum or above the clavicle near its external articulation

A large bulging should be palpated with the palm of the hand and fingers, the latter being placed in the intercostal spaces The patient is instructed to breathe deeply and then to cough. If the expansion is limited over that area, the bulging is most likely caused by an effusion. If the expansion is of greater extent, compensatory emphysema or some condition of the chest other than that of the lung or pleura must be sought A hypertrophied heart can be *detected by its pulsation, etc.* (SEE: p 396).

Depressions: Depressions are to be investigated in order to note whether they are actual or only *apparent,* because of an adjacent bony prominence. A very prominent clavicle will cause apparent supra- and infraclavicular depressions; the same holds true of the soft parts adjacent to prominent ribs, sternum, scapula or spine. A depression should be gently palpated with one or two fingers so as to note the resistance of its floor. Greatly increased or diminished resistance is of pathologic importance; if the resistance of the depressions is equal to that of the adjacent



Fig 6—Position of examiner's fingers for detecting expansile pulsation

parts of the thorax, it is most likely a congenital malformation The expansion and fremitus of such depressions should be studied further.

Pulsations (SEE p 396) The only pulsation palpable upon a normal thorax is the apex beat (fifth intercostal space *about 1 cm to the left of the midclavic*ular line). If a pulsation is visible in any other part of the chest, it should be palpated so as to *determine its extent* and character

Technic: Place the palm of one hand over the pulsating area and observe which part of the hand is being most forcibly struck; this indicates the

area of greatest intensity. Then place *the tips of the first two* or three fingers over that area and note the force, rhythm, rapidity and character. Pulsations may occur in the neck, suprasternal notch, above the clavicles, in the second and third interspaces on either side of the sternum, or in *any part of* the chest. A linear pulsation is produced by an artery or vein. A circumscribed heaving or wavy impulse may be caused by an exposed auricle or displaced ventricle. An expansile pulsation is caused by an aneurysm; this should be confirmed by other signs which will be pointed out later.

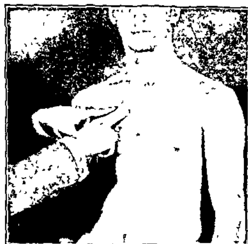


Fig. 7—Determining expansile pulsation

A *concentrated impulse* which gradually shades off into a wavy undulating motion as it leaves the center, much like the wave circles caused by a pebble thrown in the water, is caused by a pulsating empyema or by the heart violently beating against some kind of encapsulated fluid.

Expansile pulsation is best determined by bunching all the finger tips of one hand as if to grasp a small object, and placing them thus over the pulsating area; if it be expansile, the finger tips will be gently but rhythmically forced apart. Expansile pulsation may also be determined by placing the index finger of each hand at the margins of the mass; separation of the fingers by the mass denotes expansile pulsation.

Palpation alone is not a very trustworthy method for determining the actual size, shape and symmetry of the chest. Its greatest value in this direction is to confirm inspection and mensuration, the latter method being practically an instrumental palpation. In the absence of measuring instruments a general idea of the comparative size of either half of the chest may be obtained by noting how many fingerbreadths each side measures. Local irregularities, whether they be depressions or elevations, should always be palpated in order to determine their actual size and consistency.

2. *Signs That Can Be Elicited Only by Palpation*

The following can be determined only by palpation:

(*a*) The condition of the skin as to temperature, moisture, edema and certain skin reflexes. (*b*) The elicitation of pain and tenderness, precise location, distribution and degree. (*c*) The position of the trachea. (*d*) The size, consistency, mobility, and condition of the glands and organs (*e*) The presence or absence of resistance. (*f*) Tactile fremitus, vocal, rhonchal, tussive, friction and thrills (*g*) The pulse (*h*) Visible pulsation. (*i*) Study of the cardiac impulse.

(*a*) **Condition of the Skin:** ***Temperature:*** While palpation for temperature is, of course, inexact and not especially valuable, it is well, nevertheless, to cultivate the "thermic touch," because

the clinical thermometer may be broken or not at hand when it is most needed The temperature of the body can be approximately estimated, the hands of the examiner being neither too cold nor too warm, by placing the palm of the palpating hand successively upon the forehead, the abdomen and in the axilla of the patient. If the local temperature is required, the part to be examined should be palpated first and then compared with the corresponding part on the opposite side. It is best to employ the same hand for both sides of the body, first one side then the other being palpated alternately.

Undue heat of the entire surface is due to fever, to excessively warm covering, or to contact with or exposure to heat. Local increase of temperature, if not caused by being in contact with some hot object or exposure to heat, may be due to inflammation, new growth or an acute abscess

General coldness of the entire surface is caused by chills and rigor, cyanosis, poor capillary circulation, exposure to cold, and occurs during convalescence from an acute febrile disease such as pneumonia or typhoid fever, or may be due to shock and dissolution Local coldness may be caused by thrombosis or emboli, vasomotor spasm, paralysis of a certain part, and exposure to cold.

Moisture: Moisture of the skin is readily recognized by the sense of touch; general moisture of the surface, if not caused by immersion, may be due to external conditions, overheated room, hot and humid atmosphere, very heavy bedclothing, etc. The crisis of several diseases is ushered in by profuse perspiration. Malaria, septicemia, and certain stages of pulmonary tuberculosis will cause generalized perspiration. Certain nervous conditions, vasomotor reflexes, excitement, fear, laborious exercise, and the use of certain drugs may produce sweating. Local moisture may be caused by some nerve phenomenon Cold, clammy sweats are noticed in cases of hysteria, neurasthenia, exhaustion, poisoning by certain drugs and before death.

Edema: Generalized edema is usually due to cardiac or renal insufficiency; localized edema of the chest is rare, unless it is caused by some adjacent inflammation, or is postural Angioneurotic edema may occur upon the chest wall as well as upon any other surface of the body. *Localized superficial emphysema* may be mistaken for edema. The former condition occurs as a result of a punctured wound in the lungs, spontaneous pneumothorax, pneumoperitoneum and pneumothorax artificially produced, causing air to escape into the subcutaneous tissue and give rise to localized "doughy" swellings. The skin does not pit on pressure, and on palpation gives the sensation of crackling or that of a rubber bag nearly filled with air. On auscultation, when the stethoscope is pressed firmly against the mass and the patient is instructed to move his muscles, a peculiar crackling sound can be heard.

Skin Reflexes: A line drawn across the chest with a thin object will cause a momentary anemia, which is soon followed by hyperemia. This is a normal vasomotor reaction. A white line that persists for two or three minutes before hyperemia sets in, is believed by Sergent to be an indication of adrenal insufficiency (*Sergent's line*).

Pilomotor reflex (Cohen) is brought out by irritating the skin with a coin or other object.

(*b*) **Pain and Tenderness:** These may be elicited by gently palpating the overlying surface. All inflamed areas are painful to touch. Pain may also be elicited by palpating over an inflamed nerve or its distribution in a muscle or the skin. Tenderness to palpation may be caused by deep-seated inflammatory conditions. Pain and tenderness may indicate an aneurysm, broken rib, periostitis, disease of the soft parts, pleurisy, intercostal neuralgia, herpes zoster, radiculitis, disease of the lung, myocarditis, angina pectoris, sternal tenderness, etc. Referred pain and tenderness in the chest and over the sternum may arise from abdominal inflammatory disease and diaphragmatic inflammation.

(*c*) **Position of the Trachea:** Normally, the trachea is situated in the center of the neck corresponding to the midsternal line; it descends into the chest in that position, and can be felt in the suprasternal notch midway between the inner edges of both sternocleidomastoid muscles In chronic tuberculosis and fibroid phthisis the trachea is pulled toward the affected side. The trachea may be pushed toward the normal side by an extensive pleural effusion or a pneumothorax, and it may also be displaced to either side by an aneurysm, mediastinal tumor or by a spinal deformity.

Technic: The examiner should stand in front of the patient and gently fit the inner surfaces of both thumbs or index fingers simultaneously between the trachea and its adjacent sternocleidomastoid muscle. The side which exhibits a smaller space between the trachea and its adjacent muscle is recognized as the side towards which the trachea is drawn.

(*d*) **The Glands:** In the normal individual the superficial glands of the body are so small that they cannot be palpated. There are, however, various diseases that produce glandular enlargement. The disease may be one that affects a group of glands *per se* for example as in lymphatic leukemia, Hodgkin's disease, or glandular tuberculosis, or some gland may become enlarged secondary to disease elsewhere in the body, as in syphilis, malignancy, tularemia and various other conditions, local or general. When the glands are palpable the following points should be noted: Size; consistency, degree of mobility, tenderness and topographic distribution (also see Index, under Glands).

(*e*) **Resistance:** Normally, the various areas of the chest have a definite degree of resistance. *Increased resistance* of a portion of the chest indicates an altered condition of its underlying structures.

The resistance in the intercostal spaces is increased over a solid tumor, consolidation of the lung, a dense pleural effusion, chronic emphysema and local inflammatory conditions of the skin or muscle, elephantiasis and a very tense edema. In early cases of pulmonary tuberculosis, often even in the incipient stage, a certain amount of resistance (muscle spasm) can be detected in the interspaces overlying the affected parts. It is probably nature's method of "splinting" the affected part, an analogy to what is seen in the abdomen in acute appendicitis.

Diminished resistance is found over slight edema of the chest wall, and is recognized by its peculiar "doughy" feel. In the early stages of emaciation, the skin becomes loose and the muscles flabby. Muscular atrophy due either to deep-seated disease or paralysis will cause lessened resistance.

A cavity in the lung, if superficial, will cause diminished resistance as will also fluctuating tumors, lipomata, aneurysm, and small round-cell sarcoma. If a portion of a rib or muscle has been removed surgically or by an accident, the soft parts overlying this will give



Fig 8—The hands are crossed in order to "check up" fremitus perceived by palpating with uncrossed hands

rise to diminished resistance. Fluctuation is elicited in the presence of encapsulated fluid

(*f*) Fremitus: Fremitus is the term applied to vibratory tremors transmitted through the chest wall to the palpating hand. The varieties of fremitus are:

1 Vocal or tactile fremitus
2. Friction fremitus.
3 Rhonchal or bronchial fremitus.
4 Succussion or cavernous fremitus.
5 Tussive fremitus.
6 Thrills

1. ***Vocal or Tactile Fremitus:*** All varieties of fremitus must be felt, hence all are in reality "tactile" When the term "tactile fremitus" is employed, however, it usually denotes vocal fremitus

Vocal fremitus is the sensation caused by vibratory tremors transmitted to the palpating hand during talking, crying, screaming and singing. It is produced by the vibrations of the vocal cords which set into motion the entire column of air in the respiratory apparatus. These vibrations are in turn transmitted to the surface of the chest by the pulmonary structures and adjacent tissue. During vocal exercise vocal fremitus is always felt over the entire normal chest where the lungs are superficial, but in the same individual its intensity varies in different regions of the chest and it may vary in corresponding areas of different normal persons.

Vocal Fremitus in the Normal Chest: The intensity of the vocal fremitus normally depends upon: (*a*) The pitch of the voice; (*b*) the thickness and resilience of the chest wall; (*c*) the diameter of the bronchus and its proximity to the surface; (*d*) the dis-



Fig. 9—Palpating apices of lungs.

tance of the part under examination from the larynx, and (*e*) the amount of air in the respiratory tract.

(*a*) *The Pitch of the Voice:* The lower the pitch of the voice the greater is the intensity of the fremitus, and *vice versa*, because the vibrations of vocal



Fig 10—Palpating the upper posterior aspect of the chest for tactile fremitus

cords producing low pitched tones are much larger, and are carried out with greater force than the vibrations of vocal cords producing high pitched tones; just as in string (musical) instruments, the vibrations of the lower strings are much more perceptible than those of the upper strings, the former being fewer in number in a given time. In the same manner, in the human voice the difference in tone causes variations in the intensity of the vocal fremitus; everything being equal, vocal fremitus is very distinct in those having a bass voice and feeble in high sopranos

(*b*) *The Thickness and Resilience of the Chest Wall:* The thicker the chest wall, the less distinct is the fremitus, for, the vibrations having to traverse a greater distance, the acuteness of the fremitus is lost in transit. Everything being equal, the greater the resilience of the chest wall, the greater the fremitus. For this reason the fremitus is fainter over a fat chest than over a muscular one of the same size.

(*c*) *The diameter of the bronchus* and its proximity to the surface: The greater the diameter of the bronchus, the more distinct is the fremitus, because of the presence of a greater volume of air capable of being set into vibration. The nearer the bronchus to the surface of the chest, the greater the fremitus, because there is less tissue to



Fig 11—Hypothenar palpation for fremitus

interfere with the transmission of the vibrations

(*d*) *The Distance of the Part Under Examination from the Larynx:* The greater the distance, the more feeble the fremitus. That accounts for the fremitus

being greater in the upper part of the chest than in the lower.

(*e*) *The Amount of Air in the Respiratory Tract*· The greater the volume of tidal air circulating in the respiratory tract, the greater the fremitus. Vocal fremitus is more distinct when the patient speaks during inspiration than when he speaks during expiration.



Fig 12—Ulnar palpation to elicit regional tactile fremitus

Method of Palpation for Vocal Fremitus: *Technic*. The patient's chest must be bared of all clothing and he should be made to feel at ease.

The examiner assumes a position in front of the part to be examined. The palm of the hand is applied to the part under examination, and the patient is instructed to say "ninety-nine, ninety-nine, ninety-nine," or "one, two, three" Any sound that will produce the desired vibrations will do The first part of the chest to be examined is the left infra-axillary region; this region acts as a standard for the individual's normal fremitus. In the normal left lung the infraclavicular region may also be taken as a standard for tactile fremitus.

Next, the examiner places both hands *lightly but evenly* on the upper anterior part of the chest, the right hand upon the left chest and the left hand upon the right chest, while the patient utters in a deep low voice a stock phrase, "ninety-nine, ninety-nine," or "one, two, three." In case of doubt the examiner may cross his hands, so that his left hand will rest upon the patient's left chest, and the right hand upon the right chest. Another method is to use only one hand, the more sensitive of the two; while patient speaks, examiner palpates, first on one side, then on the other



Fig. 13—Hypothenar palpation for fremitus.

The most important step in the technic is to palpate the exact corresponding parts on both sides

The technic for palpating the supraclavicular regions for tactile fremitus is similar to that employed for respiratory

movements, *i. e.,* the finger tips resting above the clavicles, the examiner standing in front or behind the patient.

Posteriorly: The patient stoops slightly, his arms are held somewhat in front of him, the elbows just a little to the inside of the anterior axillary line. This position separates the scapulae, but does not put the back muscles on the stretch. The procedure employed for palpating the anterior chest wall is here repeated. Both supra- and infraclavicular regions are thus carefully palpated.

Ulnar Palpation: Many clinicians prefer the use of the ulnar surfaces of both hands, particularly to determine vocal fremitus in the interscapular regions and also to localize fremitus in the various interspaces. For interscapular palpation both hands are used simultaneously, the ulnar surface of the right hand is placed upon the right interscapular region and that of the left hand on the left interscapular region. To localize intercostal vocal fremitus, the ulnar surface of one hand only is used.

Variations of Tactile Fremitus in the Normal Chest: Generally speaking and all conditions being equal, vocal fremitus is more distinct in thin-chested individuals than in the stout, in the muscular chest rather than in the fat, flabby chest; in the male more than in the female or child; in the upper anterior part of the chest rather than in the lower and posterior aspect (the interscapular regions excepted), and on the right side more than on the left

REGIONAL VARIATIONS OF VOCAL FREMITUS IN THE NORMAL CHEST

Supraclavicular Regions
(Above Clavicles)

RIGHT	LEFT
Somewhat increased	Not quite so pronounced as on the right

Infraclavicular Regions
(Clavicles to Third Ribs)

RIGHT	LEFT
Very strong in second and third interspaces, particularly so in its inner half. Influenced no doubt by the size and position of the bronchi, and a slight increase in the density and size of the right lung.	*Quite strong, but somewhat* less marked than over the corresponding region on the right side, because the left bronchus is smaller, and joins the trachea at a more acute angle. The proximity of the esophagus and aorta also tend to diminish the force of transmission. Standard fremitus for the individual

Mammary Regions
(Third to Sixth Ribs)

RIGHT	LEFT
Vocal fremitus weak from third to sixth ribs because of large pectoral muscles and breast, also because of its distance from the large bronchus; the underlying liver also acts as a buffer.	Vocal fremitus weak as over the corresponding region on the right side because of pectoral muscles, mammae and heart

Inframammary Regions

(Sixth Ribs to Base of Chest)

RIGHT

No vocal fremitus is felt in this region during ordinary respiration. Faint fremitus may be felt in the sixth intercostal space when the patient speaks during forced inspiration.

LEFT

No vocal fremitus is felt in this region, excepting when speaking during deep inspiration

Superior Axillary

(Axilla to Sixth Ribs)

RIGHT

Very distinct, particularly in its upper part, and somewhat more perceptible than over the corresponding region on the opposite side

LEFT

Distinct, uncomplicated vocal fremitus which acts as a standard for the individual.

Inferior Axillary

(Sixth Ribs to Base of Chest)

RIGHT

Weak vocal fremitus.

LEFT

Weak vocal fremitus

Supraspinous Regions

(Above the Spines of Scapulae)

RIGHT

Distinct vocal fremitus, more distinct on this side than in the corresponding region on the left; fremitus is stronger near the spine.

LEFT

Fairly distinct, but not as intense as on the right side.

Scapular Regions

(Area Occupied by the Scapula)

RIGHT

Very weak vocal fremitus because of the scapula

LEFT

Very weak vocal fremitus because of the scapula

Interscapular Regions

(Area Lying Between Each Scapula and the Spinal Column)

RIGHT

Very intense vocal fremitus, because of the hilum of the lung.

LEFT

Quite intense vocal fremitus, because of the hilum of the lung Not quite as intense as on the right side because of the esophagus and the aorta

Infrascapular Regions

(Below the Scapula, Eighth Dorsal Spine to Base)

RIGHT

Weak vocal fremitus.

LEFT

Weak vocal fremitus.

movements, *i. e.*, the finger tips resting above the clavicles, the examiner standing in front or behind the patient.

Posteriorly: The patient stoops slightly, his arms are held somewhat in front of him, the elbows just a little to the inside of the anterior axillary line. This position separates the scapulae, but does not put the back muscles on the stretch. The procedure employed for palpating the anterior chest wall is here repeated Both supra- and infraclavicular regions are thus carefully palpated

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(Third to Sixth Ribs)

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Vocal fremitus weak from third to sixth ribs because of large pectoral muscles and breast, also because of its distance from the large bronchus, the underlying liver also acts as a buffer.	Vocal fremitus weak as over the corresponding region on the right side because of pectoral muscles, mammae and heart

vibrations produced must necessarily be great; the fact that the cavity is superficial also causes some atrophy of the overlying chest muscles, hence a shorter distance to travel and increased vibrations which are more superficial, must cause increased tactile fremitus

8. *Dilated Bronchus* (bronchiectasis): If a bronchus of normal caliber produces greater tactile fremitus than does the vesicular substance it follows that a bronchus, everything being equal, with a greater caliber must necessarily produce increased tactile fremitus (when free from secretions and superficially situated).

9. *Compensatory Emphysema* This condition should not be confounded with chronic emphysema. In compensatory emphysema the tactile fremitus is increased because there is more air in that particular part of the lung, which compensates for a lack of it in some other portion. More air in the alveoli causes increased tension of their walls and, consequently, when the air is set in motion, it will produce greater vibrations, which are readily transmitted by the tense and elastic vesicular walls The bronchioles, also being under tension, thus aid in producing increased tactile fremitus

10 *Partially Compressed Lung* This may be found adjacent to a pleural effusion, a hydropericardium, or a solid tumor The increase in the tactile fremitus results from the fact that the lung is under greater tension

11. *Resonating Chamber* It has been pointed out by Drs Chas Montgomery and LeRoy Adams that tactile fremitus, because it depends largely upon *pitch*, is often influenced by a resonating chamber. Such a chamber may be formed in the lung as a result of consolidation, while a relaxed lung may act as a nonresonating chamber.

B. *Diminished Vocal Fremitus* To determine whether in a given case the vocal fremitus is diminished, one must first form an idea of the normal fremitus for that particular individual, because, as has already been mentioned, a person having a thick fleshy chest wall or a thin high-pitched voice will naturally produce weak vocal fremitus.

Pathologically, weak vocal fremitus is caused in one of two ways: First, by any condition which will interfere with setting into vibration the air contained in the respiratory tract, Second, by conditions which will so alter the transmitting medium as to prevent the transmission of vibrations produced within the lungs to the external surface of the chest wall.

I. *Conditions which interfere with the air vibrations in the respiratory tract* and thus cause diminished vocal fremitus are·

(*a*) Partial paralysis of the vocal cords, laryngitis, or any other abnormal state of the larynx interfering with the vibrations of the cords.

(*b*) Partial compression of the trachea or a bronchus by an aneurysm, by a solid tumor, by enlarged mediastinal glands, or by an abscess

(*c*) Generalized bronchitis, by causing an inflammation of the inner lining of the bronchi, thus diminishing their caliber and elasticity

(*d*) Chronic emphysema The vocal fremitus is diminished in this condition because the whole respiratory tract is overfilled with air to such an extent as to cause a definite loss of elasticity of the vesicles and smaller bronchioles; and very little air is exchanged in the vesicu-

Vocal Fremitus in the Abnormal Chest: Pathologically, vocal fremitus may be: A. Increased. B. Diminished C. Absent.

A. *Increased Vocal Fremitus.* It has been pointed out above that vocal fremitus is caused by setting into vibration the column of air contained within the respiratory tract; the perception of this vibration by the hand is modified by the transmitting medium Therefore, any condition which compels a greater amount of air to vibrate, or produces a more readily transmitting medium will cause *increased* vocal fremitus. Increased vocal fremitus is found in: (1) Consolidation of the lung; (2) fibroid thickening of the lung (fibroid phthisis) (3) infiltration of the lungs; (4) hemorrhagic infarction; (5) adhesive bands connecting the lung with the costal pleura; (6) solid tumors lying between a bronchus and the chest wall; (7) large tense-walled superficial pulmonary cavities; (8) dilated bronchus (bronchiectasis); (9) compensatory emphysema, (10) partially-compressed lung.

1. *Consolidation of the Lung* In this condition the air vesicles of the affected part are plugged with some solid substance (exudate), so that the air contained within the bronchi and bronchioles is not permitted to enter that vesicular substance, thus causing increased tension in the bronchi supplying the diseased part of the lung The combination of vibrating air under tension and a solid transmitting medium causes increased vocal fremitus

This follows the natural law, *i e*, vibrations are more readily transmitted through a solid medium than through a liquid or gaseous one Regardless of whether the consolidation of the lung is due to lobar pneumonia, bronchopneumonia, or to pulmonary tuberculosis, fremitus is increased when consolidation is present. For obvious reasons, large consolidations produce more intense vocal fremitus than do smaller ones.

2. *Fibroid Thickening of the Lung:* The vocal fremitus is increased in this condition because the lung substance is denser than in a normal lung, and having a denser medium, the transmission of the vibrations set up by the spoken voice must of necessity be greater.

3 *Infiltration of the Lungs:* When the air vesicles are partially infiltrated with a foreign substance, the normal amount of air entering them causes increased tension of the vesicular walls Some vesicles may be entirely occluded by the infiltrate. The vibrating air under tension, added to a more densely transmitting medium, causes this increase in the vocal fremitus

4. *Hemorrhagic Infarction* Blood coagulating in the vesicles will cause a similar condition to that mentioned under (3), as the condition is practically an infiltration

5 *Adhesive bands connecting the lung with the costal pleura* will act like telephone wires and thus more distinctly transmit the fremitus produced within the lung Unless this fact is borne in mind, such an adhesive band occurring in a case of pleural effusion may lead to an erroneous diagnosis

6 *Solid Tumors Lying Between a Bronchus and the Chest Wall:* The tumor being a dense medium will transmit vibrations produced within the bronchus, thereby causing increased tactile fremitus

7. *Large Superficial Pulmonary Cavities with Tense Walls and Containing Air:* In this condition where there is a large amount of air under tension, the

it will prevent vibration. (*b*) Atelectasis or collapse of the lung from any cause will also produce absence of vocal fremitus.

2. *Pleural:* The commonest causes of absence of vocal fremitus of pleural origin are: Pleural effusions, which may be serous, sanguinous, fibrinous, pus or air, will cause absence of tactile fremitus over the area of the effusion, because in most instances the lung is either floated upward and away from the effusion, or is compressed to such a degree that the feeble vibrations there produced cannot penetrate the foreign medium.

3 *Mural:* Edema of the chest wall and diffuse lipomata are among the mural causes which fail to transmit the vibrations produced by the spoken voice. This is due to the added thickness and loss of resiliency which combine to form a nontransmitting medium of the chest wall.

Tactile Vocal Fremitus: Résumé·

INCREASED TACTILE FREMITUS

Normally

1 Male
2 Adults
3 Heavy voice
4 Thin chest
5 Right infraclavicular and both interscapular regions

Pathologically

6 Consolidations
7 Bronchiectasis
8 Superficial cavities with tense walls
9 Compensatory emphysema
10 Adhesive bands stretching between lung and parietal pleura
11 Fibroid thickening of the lung
12 Infiltration of the lung
13 Partially compressed lung overlying a pleural effusion
14 Solid tumor lying between a large bronchus and the chest wall

DECREASED TACTILE FREMITUS

Normally

1. Females and children
2 Thick chest wall
3 Thin high-pitched voice
4 Over mammae, liver and scapula.

Pathologically

5. Plastic pleurisy.
6 Thickened pleura
7 Cavity partially filled with fluid
8 Chronic emphysema
9. Asthma
10 Pulmonary edema
11 Tumors partially compressing a bronchus
12 Chronic exudative bronchitis
13 Massive pneumonia when a bronchus is partially filled with exudate

ABSENT TACTILE FREMITUS

1 Occlusion of a bronchus
2 Atelectasis.
3 Hydrothorax, pyothorax, pneuomthorax or any other effusion in the pleural sacs
4. Edema and tumors of the chest wall
5 Paralysis of the vocal cords
6. Aphonia
7 Tumor or aneurysm situated between the lung and chest wall (sarcoma, carcinoma)
8 Diaphragmatic hernia or evisceration

2. *Friction Fremitus or Pleural Fremitus:* In health, during respiration the visceral and parietal layers of the pleura constantly glide over each other without producing any sound or friction, because their surfaces are perfectly smooth and lubricated In morbid states of the pleurae their surfaces become roughened by a sticky inflammatory fibrinous exudate, which causes a grating, creaking sound when the two pleural surfaces glide over each other This sound is often detected by the palpating hand as a peculiar, vibrating, jerky or grating sensation; it occurs in interrupted jerks. The intensity of the

lar structures during normal respiration. Therefore, *when the patient is instructed* to speak, he does so with an effort. The vibrations thus produced are not very strong and are poorly conducted to the vesicles by the inelastic bronchioles. The vesicular walls also having lost their elasticity act as poor vibration conductors, thus causing very weak vocal fremitus

(*e*) Massive pneumonia, when the bronchi are plugged with cheesy material, will cause diminished fremitus because of the insufficient amount of air entering the bronchi.

II. *Conditions which will alter the transmitting medium of the vocal fremitus* produced within the lung In this class of cases, the lung substance responds normally to the vibrations produced by the column of air in the respiratory tract, but is prevented from communicating its fremitus to the external surface of the thorax by some interposing medium between the lung and the palpating hand.

(*a*) Thickened pleura. This condition gives added thickness to the chest wall Before they can be perceived by the palpating hand, the vibrations produced by the spoken voice have to travel *through an added substance which* is of a different density from that of the chest wall Because of this added thickness, much of the vibration is lost in transit. The same holds true when very small pleural effusions and exudates are present.

(*b*) Superficial cavity in the lung *partially filled* with *fluid* and *having* flaccid walls produces diminished tactile fremitus because of the inelasticity of the cavity wall and because the fluid within that cavity acts as a buffer absorbing a great deal of resilience.

(*c*) Pulmonary edema. In this condition the air vesicles contain an unusual amount of secretion, because of which very little air enters the vesicles; consequently, the tactile fremitus is very weak.

(*d*) Tactile fremitus *may be decreased* over the *entire chest* in partial compression of the trachea, chronic emphysema, generalized bronchitis, partial paralysis of the vocal cords and pulmonary edema *Localized,* decreased tactile fremitus may occur over any portion of the chest wall as a result of thickened pleura, small pleural effusions, partial compression of one bronchus, massive pneumonia, superficial partially-filled cavity, tumors in the lung or upon the chest wall, aneurysm, cyst or any other foreign body displacing a portion of the lung or superimposing upon a portion of the chest wall.

C. *Absence of Vocal Fremitus* Absence of vocal fremitus over the entire thorax may be found in those who have no voice, such as untrained deaf mutes, or those suffering from complete paralysis of the vocal cords from any cause, or as a result of certain *nervous* phenomena From the standpoint of physical diagnosis, absence of vocal fremitus is *distinctly a local condition,* never at one time affecting the entire thorax. Absence of vocal fremitus is due to pathological conditions which are either pulmonary, pleural or mural

1. *Pulmonary:* (*a*) Total occlusion of a bronchus from within or without, for example, from within, by fibrous *plugs or foreign bodies obstructing* the lumen, and, from without, by solid tumors, aneurysms, abscess or enlarged mediastinal glands compressing a bronchus, thus preventing the entrance of air to the portion of the lung supplied by

it will prevent vibration. (*b*) Atelectasis or collapse of the lung from any cause will also produce absence of vocal fremitus

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friction fremitus depends upon the quality and quantity of the exudate. A small viscid exudate will produce a more intense friction rub than will a larger or thinner effusion.

To produce a friction rub it is necessary that the two pleural surfaces should be in close proximity, and touch during at least one phase of respiration The grating appears to be superficial, and it is intensified by light pressure, but may cease on forcible palpation. A friction rub is best felt at the beginning of inspiration and at the end of expiration. Deep breathing intensifies friction fremitus The fremitus ceases when the exudate is entirely absorbed or undergoes fatty degeneration, or when more fluid is thrown out between the pleural surfaces, which acts as a lubricant. Friction fremitus is usually accompanied by pain, and because of this the patient is *often able to indicate the exact location* where fremitus can be felt by the examiner.

Technic: To palpate fremitus correctly, the patient should stand or sit upright while the examiner faces him and applies his warm palm to the spot indicated by the patient; the fingers are separated to fit the intercostal spaces. The patient is directed to breathe slowly but deeply. The stitch-like pain which usually accompanies deep breathing will often produce jerky respiration, and cause the patient to lean sharply towards the affected side. Friction fremitus is not influenced by coughing, it usually appears in the lower portion of the axillary region, and is diagnostic of acute dry pleurisy previous to the appearance of an exudate.

3. ***Bronchial or Rhonchal Fremitus:*** Bronchial or rhonchal fremitus is a peculiar sensation, not unlike that caused by the purring of a cat, transmitted to the palpating hand. It occurs in conditions where a bronchus is filled with viscid secretion and its mucosa is inflamed and thickened, thus causing a narrowing of the bronchial lumen.

The air attempting to pass through the affected bronchus sets the mucus which it contains into vibration, thus causing fremitus It can usually be felt in children suffering from a disseminated bronchitis, because of the thinness of the chest wall, and the child's inability to expectorate the accumulated secretions

In adults it is usually found in asthma, diffuse catarrhal bronchitis associated with asthenia, and advanced pulmonary tuberculosis. Bronchial fremitus is distinguished from pleural fremitus by the following points:

Résumé

BRONCHIAL FREMITUS

1 Can be felt over a large area.
2 Is continuous
3 It is temporarily checked by coughing
4 Appears deep-seated
5 Is not influenced by pressure of the hand.
6 No pain

FRICTION FREMITUS

1 Can be felt over a limited area
2 *Is jerky and interrupted*
3 Is not influenced by coughing
4 Appears superficial.
5. Is influenced by pressure
6 Pain present

4. ***Succussion or Cavernous Fremitus:*** Succussion or cavernous fremitus is a peculiar, fine sensation resembling the bursting of numerous, very small bubbles, or the gentle splashings of calm water against the shore as it is heard on a still night This condition usually occurs in large superficial cavities which communicate directly with a bronchus,

and contain both air and fluid. It can only be felt when the chest wall is thin and emaciated, and the cavity is situated near the surface in the upper lobe of the lung. It is intensified by deep and rapid breathing, and may disappear after cough and expectoration.

Succussion Splash. As its name indicates, this is a splashing sensation communicated to the palpating hand and brought out when the patient is shaken or shakes himself; it is found in cases of hydro- and pyopneumothorax.

5. ***Tussive Fremitus:*** By tussive fremitus is meant the palpable vibrations transmitted during coughing. It is of greatest value when examining deaf mutes, this being the only means of eliciting pectoral fremitus.

6 ***Thrills:*** These are palpable over superficial aneurysms, certain types of congenital heart disease, mitral and aortic stenosis (SEE: p. 403).

For (*g*) Study of the Pulse, (*h*) Visible Pulsation and (*i*) The Cardiac Impulse (SEE. p 402).

LOCATIONS OF THORACIC TENDERNESS AND THEIR SIGNIFICANCE

Causes	*Location*
Acute Pericarditis	Over the lower sternum or cardiac apical region
Acute Pleurisy	During dry stage over affected area
Aneurysm of Aortic Arch	Skin tenderness over heart, over sternocleidoid muscle or over area overlying the aneurysm.
Angina Pectoris	Often over the midsternum and precordium
Carcinoma of Ribs or Sternum	Over the affected area.
Contusion of Chest Wall	Over the injured part
Diaphragmatic Pleurisy	Over the insertion of diaphragm (10th rib), often in the neck and shoulder of the affected side.
Empyema	Over the seat of the pus
Fractured Rib	Over the seat of the pain and when pressure is exerted simultaneously to the sternum and the back pain denotes the seat of the fracture
Gastric Ulcer	Over the 10th rib at a point near the spine on the affected side
Herpes Zoster	Before and during the rash, along the affected intercostal nerve, near the spine, in the midaxillary region and near the sternum
Hydatid Cyst	Over the cyst
Intercostal Neuralgia	Along the course of the nerve and at points near the spine, the midaxillary region and the sternum
Mediastinal Neoplasm	Over the sternum or ribs
Neuritis	At the exit of the affected nerve from the spinal canal
Neurosis	Anywhere upon the chest or abdomen
Perinephric Abscess or Inflamed Kidney	Over the affected organ
Suprarenal Disease	Over the 11th or 12th rib near the spine on the affected side (Rogoff's sign)

CHAPTER XII

Percussion of the Respiratory System

Percussion of the thorax is the act of striking or tapping the surface of the thorax in order to elicit such sounds as are produced by setting the underlying viscera in vibration. The various sounds elicited by percussion depend upon the nature of the tissue struck, *i. e*, a solid substance when struck produces a dull or muffled sound while an air-containing one gives rise to a clear or resonant sound. The proportion of air and solids in the underlying organs determines the degree of clearness or dullness of the percussion sound.

Percussion as applied to the human body was first described by Auenbrügger, who in 1753 learned to distinguish by percussion the healthy from the diseased side in empyema. In 1761, after working on this subject for about seven years, he published his *"Inventum novum ex percussions thoracia humana ut signo abstrusos interni pectoris morbos detegendi."* Very little attention was paid to this work until 1808, shortly before Auenbrügger's death, when Corvisart, body physician to Napoleon the First, published the first French translation of the "Inventum novum" Corvisart also extended the application of percussion to the diagnosis of cardiac disease and aortic aneurysm. Piorry, of France, and Skoda, of Vienna, deserve credit for the most important advances in the study of percussion Piorry invented the pleximeter in 1826, and was the first to practice percussion of the abdomen. Skoda traced the qualities of the percussion sound to their physical causes, and added an exhaustive study on tympanitic sounds. Such men as Wintrich, Traube, Biermer, Geigel, Gerhardt, Neil, Welche, Sansom and Flint, all did much to advance the art of percussion. The percussion hammer was invented by Wintrich, in 1841.

Properties of the Percussion Sound

The properties of the percussion sound are based upon the classification of the musical tone. We recognize four attributes in addition to the sense of resistance:

I. Quality or timbre
II. Intensity or loudness
III. Pitch.
IV. Duration.
V. Sense of resistance.

I. Quality or Timbre

Quality or timbre, which depends upon the presence or absence of overtones is that attribute of sound which gives it its own inherent characteristics, and readily distinguishes it from other sounds of like pitch. One can easily distinguish the sounds elicited from a violin from those of a violoncello, by the different qualities of their respective tones, no matter what their pitch may be. By *quality* we mean the *kind* of sound. The two extremes of quality recognized in percussion are: (I) *Clearness,* the quality of air-containing tissue, and (II) *flatness,* the quality of airless tissue.

Between clearness and flatness there are a number of gradations in the quality of the percussion sound. These gradations depend upon the degree of admixture of airless and air-containing tissue

They are: Tympany; vesiculoresonance, hyperresonance; exaggerated resonance; resonance; impaired resonance; relative dullness; dullness; and flatness.

Clearness is further subdivided into two distinct qualities: (*a*) *Resonance,* and (*b*) *tympany*

(*a*) *Resonance* (normal lung resonance)· This term is applied to the sound elicited by percussion over normal lung substance, and is best demonstrated in the left axillary and left infraclavicular regions of normal subjects.

When normal lung tissue is percussed outside of the body (post-mortem), a *tympanitic* note is elicited; while percussion of normal lung through the chest wall elicits a "lung resonance" note. The reason the note differs in the two instances, though a similar lung is percussed, is explainable thus: In the one instance, when the lung is outside of the body, the percussion stroke sets into vibration relaxed lung substance only, *i e.*, small vesicles filled with air; therefore, a tympanitic sound is produced In the other instance, the lung within the chest, the percussion stroke sets into vibration not only the lung substance but also the parietal pleura, ribs, muscle, subcutaneous fat and skin, the latter structures being "airless," will naturally cause a dull sound, but the admixture of tympanitic lung resonance with the mural dullness produces "normal lung resonance"

It will be seen, therefore, that lung resonance depends upon several factors, and that a change in character of any one of these contributing factors will produce a distinct alteration in the quality of the *normal vesicular resonance*

In health, the normal vesicular resonance is not necessarily the same in all persons, nor in all areas of the chest in the same person. The modifying factors are as follows:

1. *Thickness of the Chest Wall:* A thick chest wall means a greater amount of airless tissue, consequently a resonance not quite so clear, and *vice versa* If the thickness of the chest wall is due to compact muscular tissue, the resonance will not be much altered, but if it be due to inelastic adipose tissue, a muffled sound is quite perceptible. A very thin and emaciated chest, particularly when the skin is stretched tightly over the ribs, gives rise to a clearer sound than normal, because of the resilience due to tenseness and the lack of a normal quantity of airless tissue.

2 *Resilience of the Chest Wall:* A chest wall which is very resilient acts as a resonator, and does not contribute as great a detoning factor as does a normal chest wall. Normally the note elicited over the sternum is clear, as the bone acts as a good resonator. Hyperresonance is also elicited over the chests of children because their chest walls are more resilient than those of adults and also because their lungs are in a state of hypertension. In aged persons a peculiar "wooden sound" is elicited, due to the ossification of the chest wall (non-resilience) and also because of a relaxed condition of the lungs.

3. *Amount of Air in the Respiratory Tract.* This has a decided influence on vesicular resonance, the resonance being clearer during inspiration than during expiration

4. *Presence of Adjacent Organs:* This quite perceptibly modifies the vesicular resonance. An airless organ like the liver or the heart, adjacent to the portion of the lung percussed, will impart a certain amount of dullness causing a

lesser degree of resonance—known as impaired resonance—because the solid organ acts as a buffer. An air-containing organ like the stomach or colon encroaching upon lung tissue will impart an added degree of clearness to that portion of the lung. Such sound is elicited normally over the base of the left lung anteriorly, and is known as vesiculotympany, or *skodaic resonance.*

(*b*) ***Tympany:*** Tympanitic or drum-like sounds are never elicited over the normal chest; their presence in the chest indicates a collection of air in the lung or in the pleural cavity Tympany is normally elicited over the stomach, colon and inflated bowel, it may also be produced by percussing over the larynx We speak of two subvarieties of tympany, namely: (1) *Open tympany,* and (2) *closed tympany.*

1 *Open Tympany*· This is elicited over large collections of air in direct communication with the outside, *i e*, large cavities in the lungs communicating through a direct opening with a bronchus. This sound can be produced by percussing over the cheek while the mouth is held open.

2. *Closed Tympany* This is a fuller sound, and is obtainable by percussing over a collection of air not in direct communication with the outside, as over the stomach, and over a large lung cavity which has no ready communication. This sound may be elicited by percussing over the cheek, the mouth being inflated and the lips closed.

Flatness and Dullness: These non-resonant qualities are obtained by percussing over airless tissue.

(*a*) ***Flatness:*** This is recognized as a greater degree of dullness, and is never found in the normal chest Its type is obtainable by percussing over the thigh or other skeletal muscles.

(*b*) ***Dullness:*** This is normally obtained by percussing over those portions of the liver, heart and spleen which are uncovered by lung tissue; the parts covered by lung give rise to *relative dullness.* No sound other than vesicular resonance should be obtained over normal lung tissue The presence of flatness, dullness or a modification thereof indicates a pathologic condition such as large pleural effusion, consolidation of the lung, thickened pleura or a solid tumor, or some other airless medium intervening between the lung and chest wall.

Résumé: The first attribute, *quality,* deals with two extremes of sound, *i e.,* *clearness* and *flatness,* and their many intermediate variations depending upon the proportion of air and solids in that tissue.

I Clear Sounds (See Fig. 1, 1-2-3-4-5)

1. Tympany. The clearest of all sounds (open and closed) obtainable over trachea, pneumothorax, lung cavity, stomach and inflated bowel
2. Vesiculotympany An admixture of vesicular and tympanitic sounds as in Traube's semilunar space, and over relaxed lung.
3. Hyperresonance. Clearer than ordinary vesicular resonance, but not as clear as tympany, elicited over an emphysematous lung
4. Exaggerated Resonance. Not quite as clear as hyperresonance, but a little clearer than normal vesicular resonance, and having all the characteristics of the latter, obtained over small areas of compensatory emphysema, also the normal note of a child's chest
5. Vesicular Resonance or Normal Lung Note: The sound obtained by percussing over lungs in the normal chest

II. Dull Sounds (See Fig 1, 6-7-8-9).

6 Impaired Resonance: Resonance not so very clear, being somewhat muffled by a small degree of dullness, found in cases presenting very small consolidations, small infiltrations of the lung, lung borders adjacent to a solid organ, and over slightly thickened pleura.

7. Relative Dullness: An admixture of dullness and resonance, the dull sound being quite perceptible This is met with in cases of small consolidation, thick pleura, or over solid organs covered by normal lung tissue.

8 Dullness: Muffled sound nearly devoid of resonance, which may be elicited by percussing over solid organs adjacent to air-containing tissue, as over the liver, heart, or spleen, and over consolidation of the lung, small pleural effusion, solid tumor and small empyemata

9 Flatness or Dead Sound Absolutely devoid of resonance. In the chest it may be obtained when percussing over a very large pleural effusion, a collapsed lung, a large aneurysm or a very large solid tumor.

II. Intensity or Loudness

The second attribute of sound is *intensity* or *loudness; it depends entirely* upon quality. The clearer the quality, the greater the intensity, and *vice versa* Therefore, a clear sound has great intensity and a dull sound little intensity, each intermediate step between clearness and dullness possessing a proportionate degree of intensity.

The intensity of the percussion sounds may be influenced by the following conditions:

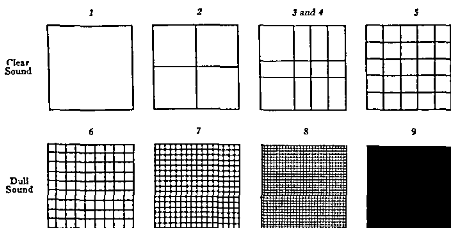


Fig 1—Résumé of sound qualities

1. **The Force of the Percussion Stroke:** The greater the force, that is to say, the stronger the blow upon the chest wall, everything being equal, the greater will be the intensity; since a greater quantity of resonant tissue is made to vibrate, therefore a greater amplitude of vibrations follows

2. **Thickness of the Chest Wall:** The thicker the chest wall, the less marked the intensity, because over a thick chest wall a duller sound is elicited than over a thin chest wall, everything else being equal

3 **The Proximity of the Part to the Percussion Finger:** The nearer the lung to the percussing finger, the louder the sound produced by percussion.

4. The Amount of Air-containing Tissue Set into Vibration: The more air set in vibration by the percussion stroke, the greater will be the intensity.

Résumé: Intensity means loudness The percussion sounds elicited over air-containing tissue are resonant in quality and loud in intensity. Airless tissue produces dull sounds, which are low in intensity. Intensity depends upon the clearness of the sound, and is modified by the force of the percussion stroke, the thickness of the chest wall, the proximity of the part to the percussion finger, and the amount of air set into vibration.

III. Pitch

The third attribute of the percussion sound is termed *pitch;* it depends upon the degree of elevation or depression of *the note. It is the shrillness of the tone,* depending upon the length and rapidity of vibrations. The shorter and more rapid the vibrations, the higher the pitch. Of all the attributes of the percussion sound, pitch is the hardest to master, as it requires a somewhat musical ear.

Pitch is the most important of the attributes in distinguishing fine variations of sound which are used in outlining certain organs, or for detecting minute pathological conditions of the lung. It is, therefore, of great importance to the student to devote much time to the practice of percussion and thoroughly familiarize himself with the different attributes of sound.

The pitch, as a rule, is *high* in dull notes and *low* in clear notes. The clearer the sound the lower the pitch, or conversely *the duller the sound the higher* the pitch.

The pitch over a normal lung is low; *increased tension in the lung causes a* higher pitch; relaxed lung produces a lower pitch.

Normally, the pitch is influenced by: (1) The thickness of the chest wall. The thicker the chest wall, the higher the pitch. (2) The amount of air in the lungs. The more air in the lungs during percussion, the lower the pitch.

Pathologically, the pitch is influenced by: (*a*) The condition of the lungs, consolidated, relaxed or cavitated (see *pitch* in pathologic percussion); (*b*) the condition of the pleura, and (*c*) the condition of the chest wall.

Pitch of Tympanitic Sounds: The pitch is higher in open than in closed tympany.

Open Tympany: The height of the pitch depends upon: (*a*) The size of the communicating opening; the larger *the opening, the higher the pitch.* (*b*) The volume of air contained in the cavity; the more air, the lower the pitch. (*c*) *The tension of the cavity walls;* the greater the tension, the higher the pitch

In cylindrical cavities communicating with the outside air, the pitch is heightened in proportion to the length of the *tube*

Closed Tympany: The pitch here also depends upon the tension of the *cavity walls and the amount of air in the* cavity. Greater tension of the cavity walls causes higher pitch; more air in *the cavity causes lower pitch* A large cavity, other things being equal, produces a lower pitch than does a small one.

IV. Duration

This is the fourth attribute of sound *It depends* upon the length of the sound waves and their persistence. It is simply the length of time a percussion note can be heard. Clear sounds can be heard

longer than dull ones, therefore, the clearer the sound, the longer is its duration.

V. Sense of Resistance

During percussion over the various parts of the chest, a certain amount of resistance is perceived by the pleximeter as well as by the plexor finger. This depends upon the consistency of the tissue percussed, and bears a direct ratio to quality and pitch. The more resistant the tissue, the higher the pitch Air-containing tissues have a lower resistance than have airless structures.

Résumé:	Air-containing Tissue	Airless Tissue
Quality	Clear	Dull
Intensity	Loud	Low
Pitch	Low	High
Duration	Long	Short
Sense of resistance	Slight	Marked

Methods of Percussion

There are two methods of percussion · (*A*) The immediate; (*B*) the mediate

A. **Immediate Percussion:** Immediate or direct percussion was first practiced by Auenbrugger in 1761. It consisted of slapping the chest wall, without any interposing medium, with the palmar surface of one hand. At present several modifications of the original percussion method are employed. As a general rule, the results obtained by mediate percussion are far more trustworthy than those obtained by the immediate method Except in isolated instances, immediate percussion is not generally practiced

The methods and technic for direct or immediate percussion are:

1. ***Palmar Percussion:*** The chest wall is slapped smartly with the palm of one hand or with the palmar surface of the four fingers of one hand. The blows are delivered from the wrist at the rate of about two per second; the examiner comparing the note thus elicited with that produced on a corresponding part on the opposite side This method is at times employed in order to determine the gross differences of sound between different regions of the same side or upon opposite sides of the body, rather than to localize with any degree of accuracy lines of demarcation of organs or pathologic processes; pleural effusions and very large tumors may be recognized by this method, but it has no advantage over mediate percussion.



Fig 2—Direct or immediate percussion over a bony prominence.

2. ***Immediate Finger Percussion:*** A bony prominence, such as the clavicle, sternum or a protruding rib, is struck directly with the tips of one or two fingers, usually the index finger or the index and middle fingers. The bone is struck smartly with moderate force at the rate of two strokes per second This method is convenient, as the bony prominence acts as a pleximeter, and the results thus obtained are accurate. Immediate finger percussion is also advan-

tageously practiced for accurately outlining the heart borders. The examiner "pulls up" the soft tissues over the heart area with his left hand, the index finger of the right hand being reinforced by the middle finger, and the chest wall is struck sharply with the soft portion

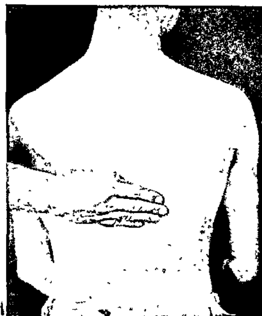


Fig 3—Technic, direct or immediate or palmar percussion

of the upper phalanx at the rate of two per second, the blows being delivered from the wrist. Percussion is started at the resonant portion of the chest at right angles to the heart, the heart border thus being gradually approached.

3. ***Flipping:*** This method is sometimes employed for superficial percussion of the chest and abdomen. The dorsal surface of one finger is constrained and then suddenly released, so as to strike the surface under examination.

4. ***Ulnar Percussion:*** This method is employed for eliciting tenderness over the gallbladder and kidney. It is a modification of "fist percussion" employed by Murphy. The patient is asked to take a deep breath and hold it. The examiner strikes a sharp blow over the upper part of the right rectus muscle, with the ulnar side of the hand. Disease of the gallbladder *is manifested by sharp pain.* This procedure should be employed over both recti muscles in order to exclude muscle pain.

B. **Mediate or Indirect Percussion:** This consists in striking the chest wall through some interposing medium. The three methods are: (1) Finger percussion; (2) plexor, pleximeter percussion; (3) combined method.

1. ***Finger Percussion:*** The interposing medium is a finger; one finger is



Fig 4—Ulnar percussion to elicit gallbladder tenderness.

laid flat upon the chest wall and it is struck with one or two fingers from the other hand. The finger receiving the blow is called the *pleximeter*; that with which the blow is struck is called the *plessor or plexor.*

2. *Plexor, Pleximeter Percussion* (instrumental): Instead of a finger, a small piece of ivory, vulcanite, or some other hard substance made to conform with the chest may be used as a pleximeter. The *pleximeter* used by Piorry consisted of a thin oval ivory plate, about 2 cm wide, with edges turned up, so that it could be held firmly by the thumb and finger. Many other shapes and sizes of pleximeters have been brought forward by clinicians; the principle of all of them, however, is similar.

The *plexor*, or hammer, was invented by Wintrich in 1841; it is a light hammer, the handle of which is made of wood or wire and the head of india rubber. Sounds are readily brought forth with these instruments. They are useful for class demonstration, or where a large number of patients have to be forcibly percussed.

Their disadvantage for general use is, in the first place, that the pleximeter cannot always be made to conform to the surface of the chest, secondly, that they produce a certain sound of their own; thirdly, one or both of the instruments may not be at hand when they are most needed; fourthly, when one becomes accustomed to their exclusive use, finger percussion becomes entirely unsatisfactory. These disadvantages do not apply to finger percussion When one once masters this he will be equally well able to use any instrument for bringing out percussion sounds.

3 *The Combined Method:* An instrumental pleximeter may be used and the finger as a plexor, or the finger may act as a pleximeter and an instrumental plexor may be used. The combined method has no advantage over either (1) or (2).

Starting Point for Percussion

As a general rule, the first areas to be percussed are the axillary or the subclavicular regions of the left side. The resonance there obtained represents the normal lung resonance for the individual. Having thus obtained the required note, general percussion is commenced. Most clinicians begin by percussing the apices of the lungs and gradually work toward the bases, anteriorly, laterally and posteriorly.

Many clinicians start percussion at the base and gradually work upward toward the apex. The same procedure is carried out anteriorly, posteriorly and laterally in the order named. Having familiarized oneself with the general note of each side, the various areas or intercostal spaces of one side are compared with those of the other side.

The rule to be remembered is this: *Percussion should always be started from a known resonant area and carried toward a suspected dull area.*

When examining for pulmonary tuberculosis, percussion should be started at the base and carried toward the apex, because the apices of the lungs are, in an adult, most frequently the first seat of a demonstrable tuberculous lesion.

When examining for lobar pneumonia or pleural effusion, percussion may be started above and carried downward toward the dull area.

When the heart is to be outlined, percussion should be started at some distance from the usual heart border, at a point well within the resonant part of the chest, the heart is thus approached in a line parallel to its border.

Technic for Mediate Percussion

In order to elicit the exact notes obtainable by percussion over a certain

area, one must be painstakingly accurate in learning the proper technic required for its performance.

Percussion consists of two phases, an active one, *striking*, and a passive one, *listening*. It must be remembered that the results of percussion depend not so much upon the striking of the chest wall, *as upon the sounds produced thereby*, and hence one must concentrate his entire attention upon the sound elicited and not upon the act. The act of percussion must be carried out in a definite way so as to elicit the correct note, and the proper performance should, with constant practice, become a subconscious act requiring no mental effort. Stress is here laid on the technic of *finger percussion*, because this method is the most practical and when once mastered, the results thus obtained are accurate. The *practicability of this method is obvious*, because the examiner always has his fingers with him; they cannot be forgotten or misplaced when they are needed, and the fingers can be made to fit any surface desired; besides, there is a certain sense of resistance appreciated by both the plexor and pleximeter fingers, when percussing certain tissues, which cannot be perceived by instrumental percussion.

Rules to Observe During Percussion

It is most important for the proper performance of percussion that certain rules regarding the position of the patient and physician should be observed.

I. The patient should bare his chest in its entirety, because clothing interferes with the true chest sound, it being an added thickness of unknown acoustic properties. If an over-modest female patient objects to baring her chest, or when the room is too chilly, a thin unstarched garment may be allowed to cover the chest.

II. The patient must assume a perfectly natural and unconstrained position, whether standing, sitting or lying in bed. He should be thoroughly relaxed, so as to avoid as far as possible any degree of muscle rigidity, because *muscular rigidity interferes with the* quality and pitch of the percussion sound.

(*a*) **Standing Position:** ***Examination of the Anterior Aspect of Chest:*** The patient is to stand erect, arms hanging loosely at his sides, head in the median line, the chin should not be too much raised or lowered. Military-like erectness should be avoided

Examination of the Posterior Aspect: *The patient is to stoop slightly* forward, his arms hanging loosely and somewhat inward; this will avoid muscular strain and at the same time separate the scapulae

Examination of the Lateral Aspect: The patient should put the hand of the side to be examined upon his head and not allow it to extend at right angle with the body.

(*b*) **Sitting Posture:** The patient is to sit erect, though somewhat bent forward, the chin lowered and the arms hanging loosely at the sides. He should avoid, if possible, leaning against anything, because the object against which the patient may lean is likely to act either as a *sounding board or muffler*, thus interfering with the normal sound dissemination. An exception must be made with patients who are too weak to sit upright for any length of time, or are unable to bear the percussion stroke unsupported. An unconstrained and *thoroughly relaxed posture must* be assumed by the patient, irrespective of what his examining position may be.

The posture assumed by a sitting patient for the examination of the posterior and lateral aspects of the chest is similar to that assumed by a standing patient when these surfaces are examined. In order to detect movable dullness, the patient is to lean sharply toward the suspected side and the upper level of percussion dullness is noted on that side; the patient is then made to lean sharply toward the opposite side, and is allowed to remain in that position for several minutes, in order to allow time for gravitation, then the affected side is again percussed and the upper level of dullness is noted. A change in the level of dullness indicates the presence of free fluid. The line of dullness is higher when the patient leans toward the affected side.

(*c*) **Lying Posture:** ***Examination of the Anterior Aspect:*** The patient is to lie upon his back resting easily, his head in the median line and the hands at his sides.

Examination of the Posterior Aspect: The posterior aspect of the chest of a very sick patient is examined by turning him upon his side so that his back faces the examiner, or he may be rolled over on his abdomen Changeable dullness may be detected by first noting the upper level of dullness in the lying posture, then the patient is gently raised to the sitting or semireclining position and the upper level of dullness is again noted

Examination of Lateral Aspect: The lateral aspects may be percussed in the same position as the anterior, or the patient may be gently rolled, first upon one side, then upon the other, as the case may require.

III. The examiner must have his hands warm and free from moisture, and his fingernails, particularly of the plexor finger, should be short.

IV. The examiner should assume a natural and unconstrained position, so that he may manipulate his hands at will, without causing any strain on his wrists The part under examination must always be directly in front of him.



Fig. 5—Technic for percussion of thorax.

V. Both sides of the chest should be accurately compared The pleximeter finger should be applied to corresponding spots on the two sides If an interspace is percussed at a certain distance from the median line on one side, precisely the same location must be chosen on the opposite side. If percussion is performed during either inspiration or expiration on one side, it should be carried out during a similar act on the opposite side

VI. **Pleximeter Finger:** The index or middle finger of the left hand in a right-handed person or the corresponding fin-

area, one must be painstakingly accurate in learning the proper technic required for its performance.

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as it is less likely to produce adventitious sounds; multiplicity may cause error.

(*b*) The percussing finger should be bent at the second *joint*, the distal digit being held rigid, so that the blow can be struck perpendicularly with the rounded end of the finger nearest the palmar side.



Fig 8—Technic for percussion of supraclavicular space.

(*c*) A glancing blow should always be avoided.

(*d*) The percussion stroke should be of moderate force for ordinary percussion and administered at the rate of about *two strokes per second*.

(*e*) The plexor finger must be withdrawn as soon as it strikes; if the finger is permitted to linger after delivering the blow, the sound produced will be muffled.

(*f*) The force of the blow should be delivered entirely from *the wrist*, the elbow being held rigid.

VIII. **The Percussion Stroke:** The force to be used in percussion depends upon the apparent situation of the lesion and the region which is being percussed.

The percussion stroke may be *light*, *moderate* or *forcible*.

Light percussion should be practiced when percussing over superficial organs, as the apices of the lungs, in the examination of patients having thin chest walls, and in children.

Moderate percussion is employed for general use.

Forcible percussion is to be used when deep-seated structures are involved, or when examining patients who have very thick chest walls.

It should be noted that when a rib is percussed anteriorly, the sound is carried along that rib to its posterior position, which is about three or four intercostal spaces higher than its anterior plane.

Alteration of the Force of the Percussion Stroke: There are conditions requiring a change of the percussion stroke from medium to light, or heavy. If, because of signs obtained by inspection and palpation, a lesion in the lung is suspected and cannot be demonstrated by light percussion, the medium stroke should be employed; if this fails to reveal the presence of a lesion, then the heavy stroke should be used. A heavy percussion stroke should also be employed when outlining a solid organ covered by lung. Often a heavy stroke may not reveal any lesion while a medium or light stroke will. This phenomenon can be thus explained:

(*a*) If the lesion is superficial, a heavy percussion stroke passes through the dull area and sets the normal lung beyond it into vibration; thus a clear sound is elicited.

(*b*) If the lesion is of medium depth, a light stroke may not reach it and a heavy stroke will pass through it. As a

ger of the right hand in a left-handed person is chosen as the pleximeter.

(*a*) It should be placed in an intercostal space evenly and firmly, with the palmar surface downward.

(*b*) The pleximeter finger alone should rest upon the chest wall during percussion, because if the other fingers, or any part of the hand, are allowed to rest upon the chest wall, they will interfere with chest vibrations, thereby muffling the elicited sound.



Fig. 6—Technic for percussion of axillary region.

(*c*) The pleximeter finger should exert a moderate amount of pressure, sufficient to exclude the air between it and the chest wall. An intervening column of air, no matter how small, when percussed will give rise to an abnormal sound. However, too much pressure is to be avoided, as it may cause pain.

(*d*) The same amount of pressure should be exerted on opposite sides, because when light pressure is exerted on one side and moderate or strong pressure is used upon a corresponding portion of the opposite side, the percussion note will vary, though the lung condition be the same on both sides

(*e*) When percussing the anterior and lateral aspects of the chest, the pleximeter finger is held parallel to the ribs.

(*f*) The interscapular regions, on account of the proximity of the ribs and the position of the scapulae, may be percussed by holding the pleximeter finger perpendicular to the ribs

(*g*) In the supraclavicular regions, any finger may be chosen which fits them most snugly. Some clinicians employ their thumbs as pleximeters, others their index fingers; the little finger is often the most convenient pleximeter for the supraclavicular regions.



Fig 7—Technic for percussion of the posterior aspect of chest.

VII. **The Plexor Finger:** (*a*) The plexor finger is usually the middle finger of the right hand; some clinicians employ both the index and middle fingers, striking with both fingers simultaneously. It is best to use the middle finger alone,

as it is less likely to produce adventitious sounds; multiplicity may cause error.

(*b*) The percussing finger should be bent at the second joint, the distal digit being held rigid, so that the blow can be struck perpendicularly with the rounded end of the finger nearest the palmar side.



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(*b*) If the lesion is of medium depth, a light stroke may not reach it and a heavy stroke will pass through it. As a



Fig. 9—The flexor finger upward.



Fig 10—Position of flexor finger in downward stroke



Fig 11—The percussion stroke.

general rule, however, the percussion stroke should be of medium force and delivered in a manner described before; most lesions in the lung can be reached by such a stroke, except those occurring at the apex Because of the small amount

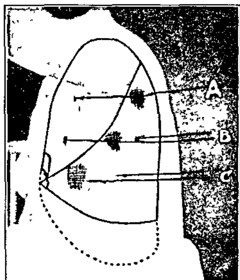


Fig 12—Force of percussion stroke required to reach lung lesions, illustrated by A, B and C

of lung in this region, the percussion stroke should be light.

(*c*) If the lesion is deep seated and the percussion stroke is very light, the vibrations thus produced do not reach the lesion, consequently a clear note is obtained.

Respiratory Percussion

This term, introduced by J M. Da Costa, is applied to percussion during the act of deep inspiration and forcible expiration, percussion in each instance being performed while the patient holds his breath

During inspiration, the note is more resonant; the lung apex is somewhat higher; the base of the lung is lower, and the lateral borders encroach more upon the sternum.

Auscultatory Percussion

This method was first described by Drs Clark and Camman, of New York, in 1840 It is especially useful for outlining such organs as the liver, spleen and heart and at times also a distended stomach and colon

Technic: The examiner places the stethoscope upon the supposed border of the organ farthest removed from the edge to be percussed, and holds it there with one hand, while with one or two fingers of the other hand he begins tapping the surface a short distance away from the supposed border, when the border of that organ is reached, a change in quality is at once perceived



Fig 13—Technic for auscultatory percussion.

It is well to observe Cabot's caution in regard to outlining an organ accurately; he moves both hands, one holding the stethoscope and the other percussing,

always keeping the hands the same distance apart, while approaching the center.

The author usually holds the bell of the stethoscope between the index and middle fingers, close to the surface, and strikes the first phalanx of the index finger; in this way a definite distance is maintained between the stethoscope and pleximeter. When a disk chest piece is used, it may be held in position with the palm of the hand while the finger is being struck.

Phonometry or wave auscultation described by Bass, in 1880, may yield fairly good results; a tuning fork is substituted for the percussing hand. The stethoscope is held over the organ to be examined, as in the method already described, and an ordinary tuning fork is set into vibration by striking it against some object; the handle of the tuning fork is set base downward upon the chest or abdomen and is rapidly moved toward the supposed organ or cavity. By this method it is at times possible to outline the superficial area of cardiac dullness, superficial consolidation of the lung, pleural effusion, superficial cavity in the lung, or to determine the size of the stomach or other superficial organ. This method may also be modified by placing the vibrating tuning fork upon the surface overlying the organ and gradually approaching it with the stethoscope. Phonometry is of doubtful value as to accuracy.

Palpatory Percussion

Palpatory percussion may be carried out by both the immediate and mediate methods. In the *immediate method* the chest wall, particularly the intercostal spaces, are struck lightly with a pushing movement by the sensitive portions of the finger tips in order to determine the resistance of the part. In the *mediate* method, numerous light glancing pushing blows are applied to the pleximeter finger, thereby bringing out the resistance of the part. It requires much practice and a delicate sense of perception to master this method. It has its greatest usefulness in mapping out organs for those physicians, particularly, whose sense of hearing is defective, or it may be employed upon individuals who, for any reason, should not be audibly percussed.

The Normal Chest

Regional Percussion

It is essential that one should be thoroughly familiar with the normal sounds elicited in the various regions so as to recognize any deviation therefrom.

Anterior Aspect: ***Supraclavicular Regions:*** *Krönig's isthmus* is a strip of resonance extending across the trapezius muscle and corresponding to the apex of the lung; contraction of this area denotes disease of the lung apex.

Technic for Eliciting Krönig's Isthmus: The examiner stands behind the patient, who sits in a chair. The first phalanx of the pleximeter finger is placed upon the inner edge of the trapezius muscle at a point corresponding to the midclavicular line; it is then gently percussed with the plexor finger; percussion is carried toward the neck, and at the point where the note changes from resonance to dullness a pencil mark is made. The percussion is then carried outward toward the acromion process, and here again, when the note changes from resonance to dullness, another pen-

cil mark is made. The distance between the two pencil marks represents the size of the isthmus, usually about the breadth of three fingers (5 cm.).

The supraclavicular regions are triangular in shape and are situated each above its respective clavicle, and contain the apex of that lung. These regions are important because manifest pulmonary tuberculosis in an adult usually makes an early appearance there. The percussion note varies somewhat in each region; a light stroke should be employed

Clavicular Regions: The clavicles act as sounding boards, increasing the resonance of the entire thoracic cavity, hence the percussion note is generally clear, and is almost tympanitic near the sternum because of its proximity to the trachea.



Fig 14—Technic for percussing Kronig's isthmus

Infraclavicular Regions: These regions being situated on either side of the sternum, occupying the space from the lower margin of the clavicle to the upper edge of the third rib, contain practically pure vesicular lung structure and its

RIGHT

Impaired resonance in outer half. Hyperresonance at inner third because of the proximity of the trachea, the right apex does not extend quite so high as, and is smaller than, the left one; the muscles covering this region are, as a rule, more developed; the superior vena cava and right subclavian artery lie more anteriorly on the right side; also the right lung contains more bronchioles, airless tissue Therefore, the percussion note is not quite so clear as on the opposite side The pitch is somewhat higher.

LEFT

Impaired resonance in outer half, but clearer than in the right. Resonance in inner third Because of the greater amount of lung in this region, and for the other reasons given, the note is somewhat clearer on this side than on the right. The percussion sound is clearer at the sternal extremities in both of these regions, because of their proximity to the trachea. The resonance diminishes as the acromion angle is approached.

enveloping pleura. The percussion note, however, differs slightly on the respective sides. A medium percussion stroke should be employed.

RIGHT

Clear vesicular lung resonance, but not quite so clear as on the left side, because of the more numerous bronchioles, and also because the right lung is supported by the liver, which acts as a buffer

LEFT

Typically clear vesicular resonance, or normal lung resonance. This region may be used as a standard for clearness for each particular individual In the second interspace, close to the sternum on both sides, the percussion sound assumes a muffled tympanitic note, due to the bifurcation of the trachea.

Mammary Regions: The situation of these regions (third to sixth ribs) and the heavy, muscular, fatty and glandular coverings greatly modify the percussion note, which presents marked differences on the two sides of the chest.

RIGHT

Vesicular resonance from the third rib to the fourth interspace, though somewhat muffled on account of the thickness of the chest wall; usually a somewhat heavier percussion stroke is required

Impaired resonance below the fourth interspace to the upper margin of the sixth rib, because of the underlying liver.

Relative dullness close to the sternum from the third to the fifth intercostal spaces, where the thin edge of the lung overlies the heart

LEFT

Impaired resonance from the third to fourth rib inside the midclavicular line because the heart is covered by lung. A very heavy percussion stroke in this area will elicit a relatively dull note.

Cardiac dullness from fourth rib to fifth interspace; below that a dull note is elicited due to the recession of the left lobe of the lung It should be remembered that relative dullness and dullness elicited on the left side are normal only when occurring to the right of the left midclavicular line

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Inframammary Regions: Situated below the sixth rib and occupying the remainder of the chest cavity, they are formed by the converging and coalescing ribs; their respective contents being at variance with each other, give rise to the following percussion sounds:

RIGHT

Dullness (due to liver) from sixth rib downward, the lowermost portion of this region may give rise to a mixture of tympany and dullness, the former caused by an inflated hepatic flexure.

LEFT

Vesiculotympany from the sixth rib to the lower margins of the ribs to the left of the midclavicular line

This region is known as Traube's semilunar space It is bounded above by the heart and lung; on the inside by the left lobe of the liver, and posteroinferiorly by the spleen It contains the cardiac end of the stomach Splenic dullness is elicited over the spleen (ninth to eleventh ribs) on forcible percussion

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Lateral Aspect: ***Supraäxillary Regions:*** These extend from the hollow of the armpit to the sixth rib and contain lung and pleura. The percussion note elicited throughout these regions on both sides is clear vesicular resonance, though it is somewhat clearer on the left side than on the

right, the former often being used as a standard for the normal lung resonance of the individual

Infraaxillary Regions (below the sixth rib).

RIGHT	LEFT
Clear vesicular resonance from sixth to seventh rib Impaired resonance from seventh to eighth rib Dullness below that, due to liver.	Clear vesicular resonance from sixth to eighth rib Vesicular tympany to the right of the median line from eighth rib downward *Relative dullness or dullness is elicited* between the posterior axillary and midaxillary lines from the ninth to the eleventh ribs due to the position of the spleen (splenic dullness)

Posterior Aspect: The percussion sound over the dorsum of the chest is duller, and the pitch higher, because of the following facts:

1. The closeness of the ribs
2. Their insertions almost directly upon another osseous structure which is not a resonator (the spine)
3. The peculiar curvature of the ribs and their heavier dorsal extremity
4. The difference in the structure of the soft parts with the addition of the scapulae

Supraspinous Fossae (above the spine of the scapulae) · The note is muffled vesicular resonance The pitch is a little higher, and the resistance somewhat greater at the right supraspinous fossa than at the left. These regions should be percussed with a heavier stroke while the patient is in the erect or in the stooping postures. Persistent dullness indicates consolidation of the apex of the lung

Scapular Regions: On account of the scapulae, the percussion sound here elicited is relatively dull

***Interscapular Regions, i e*,** the area between the scapulae from the third to the eighth dorsal spine on either side of the spinal column: Vesicular resonance is not very clear in these regions, because of their close proximity to the spine and their muscular coverings. The vesicular resonance is also *slightly* modified by the trachea, and the bronchi, which enter the lung in this region at the level of the fifth dorsal spine

Infrascapular Regions (below seventh rib) · *These regions produce the* greatest amount of vesicular resonance posteriorly

Clear vesicular resonance prevails on the *right side* from the seventh to the ninth rib, relative dullness from the ninth rib to the tenth; below the tenth rib liver dullness is elicited. *Left side,* vesiculotympany from seventh rib downward to splenic dullness

Respiratory Mobility

The base of each lung descends during inspiration and ascends during expiration *Posture* to some extent also influences the lung borders according to gravity. This is particularly noticeable when the patient turns from the recumbent posture to either side A greater descent of the diaphragm is noted on the dependent side

Complementary Spaces: The respiratory mobility of the base of the lung is noted in the following manner:

Technic: The patient stands or sits, his back toward the examiner.

1. During normal respiration, the examiner maps out by percussion the lower border of the lung and marks it with a pencil.

2. The patient is then instructed to take a very deep breath and to hold it while the examiner percusses the level to which the lung has descended and places another pencil mark.



Fig. 15—Technic for outlining the complementary spaces.

3. The patient is then instructed to exhale forcibly and arrest the act after expiration is accomplished. The examiner again percusses to find the level to which the lung has ascended and again places a pencil mark.

The space between the upper and lower pencil marks represents the respiratory mobility or complemental space. The same act is repeated on both sides of the spine.

The left lung usually descends a half-inch lower than the right. In disease of the base of the lung and also in pleural and diaphragmatic adhesions, the respiratory mobility is diminished. In large pleural affusions, pneumothorax, *hydrothorax* and pulmonary atelectasis, respiratory mobility is practically *nil*

Topographic Percussion

Percussion is the only means at our command for determining by physical examination the sizes of the various organs contained within the thoracic cavity. It is, therefore, necessary for one to be familiar with the normal size of an organ, so that he may judge it in diseased conditions, and note if a particular organ is increased or diminished in size The anatomical position of the various viscera has been mentioned in a preceding chapter.

In order to determine the exact size of the various organs or to differentiate the borders of two organs that lie adjacent, so as to know where one viscus begins and the other ends, they must be of different densities Thus, we can easily tell where the lung ends and the heart begins, but it is impossible by percussion to differentiate between heart and liver dullness, or between a pleural effusion and the liver border

Technic: To properly outline an organ, percussion should always be started from a resonant organ so as gradually to approach the nonresonant one. In this way the elevation in pitch can be noted. The pleximeter finger should be placed parallel to the supposed border of that organ.

Light percussion should be practiced at the junction of any two organs.

It is important to note that the lower border of the lungs and of the heart are one interspace higher in children than they are in adults. Thus, in children, anteriorly, the lower border of the lungs is in the fifth intercostal space, laterally

in the seventh, and posteriorly in the ninth. The apex beat is in the fourth interspace. On the other hand, in very old people the lung borders are an interspace lower than in normal young and middle-aged adults; thus the lower anterior border of the lung is in the seventh interspace, laterally in the ninth interspace, and posteriorly in the eleventh interspace. It will be noted that the relation of the base of the lungs to the ribs in the anterior, the lateral and the posterior aspects is the same at the various stages of life.

	Anterior	Lateral	Posterior
Young children	5th rib	7th rib	9th rib
Adults	6th rib	8th rib	10th rib
Old people	7th rib	9th rib	11th rib

The difference of lung and rib topography at the various ages of life is probably caused by the difference in the angles of the ribs at these ages. In children, the ribs are horizontal, and at right angle with the sternum. In young and middle-aged adults, the ribs are somewhat oblique. In old age, the ribs take a decidedly oblique course.

The Abnormal Chest

Pathologic Variations of the Percussion Sound

Normally, the only percussion sound elicited over the lungs is vesicular resonance, with slight modifications in its pitch and its intensity, depending upon the thickness of the chest wall and the proximity of other organs

Pathologically, the percussion note may vary from absolute dullness to tympany, with all their intermediate variations, depending upon the specific morbid condition of the lung, pleura and chest wall.

A. Abnormal Dullness

I. **Dullness and Flatness**: Dullness is elicited only over airless tissue adjacent to air-containing structures. If a dull note is obtained by percussing over the lung, it indicates that air-containing lung substance has been metamorphosed into an airless tissue The following conditions produce dullness:

1. ***Intrapulmonary:*** (*a*) *Consolidation of the Lungs* (the pneumonias and pulmonary tuberculosis): The air vesicles, being filled with inflammatory exudate to the exclusion of all air, are practically solid substances and hence they yield a dull percussion note The larger the consolidation, the more pronounced is the dullness, because in large consolidations the percussion stroke is unable to set the surrounding vesicular structures into vibration. The note thus elicited is, therefore, very dull, because it is not tinctured with an adjacent resonance-producing substance.

(*b*) *Pulmonary Atelectasis:* In this condition the lung is collapsed and forms airless tissue; consequently, there will be dullness on percussion.

(*c*) *New Growths in the Lung Substance:* Carcinoma, sarcoma, gumma, abscess, cyst, enlarged mediastinal or bronchial glands, and aneurysm, because of their solid consistency, and when large, will produce a dull percussion note.

(*d*) *Large Hemorrhagic Infarcts, or Gangrene of the Lung:* If superficial and before they have undergone complete necrosis and cavitation, these will produce dullness.

2. ***Extrapulmonary:*** ***Displaced solid organs,*** like transposed viscera, cause dullness in unexpected regions. Thus

heart dullness may be obtained at the fourth or fifth interspaces on the right side, and liver dullness over the lower ribs on the left side.

wall is replaced by fluid which is an airless tissue.

Fluid in the pleural sacs, when not bound down by adhesions, is *freely mov-*

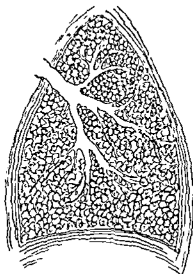


Fig 16—Normal lung, over which vesicular resonance is elicited

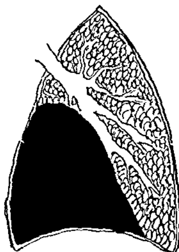


Fig. 17—Consolidation of lung yielding a dull note.

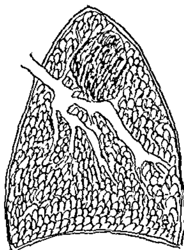


Fig 18—New growths of lung yielding relative dullness.

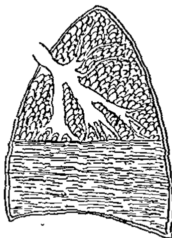


Fig 19—Pleural effusion yielding flatness.

Pleural Effusions. Hydrothorax, pyothorax, hemothorax, or any other liquid effusion in the pleural cavity, will yield absolute dullness or *flatness.* The reason for this is self-evident—the lung structure being pushed away from the chest

able, the fluid always gravitating to the dependent parts A change of the patient's posture will, in such cases, cause a change of the upper level of dullness. Under certain circumstances, the upper level of liver dullness may be shifted

and therefore be mistaken for pleural effusion; for instance, in the *sitting* or standing posture, liver dullness may be one or two intercostal spaces higher than in the recumbent position When recumbent, the liver gravitates toward the back, allowing room for the base of the lung to descend, in the sitting or standing position, the lung is supported by the liver. The diaphragm usually accommodates itself to the kind of support it receives. Grocco's sign is of value in differentiating pleural effusion from a movable liver.



Fig 20—Grocco's paravertebral triangle of dullness

Grocco's sign (paravertebral triangle of dullness) · The dullness elicited posteriorly in the presence of a pleural effusion, occurring on one side, is transmitted to a triangular area of dullness on the opposite side of the spine The apex of the triangle corresponds to the upper level of the effusion on the affected side; the base is formed by the lower level of dullness and the hypotenuse extends from apex to base. It can be elicited when the patient either stands or sits upright. The triangular area of dullness disappears when the patient lies on the affected side.

Tumors, either solid or an aneurysm, when situated at the base of the lung, will cause dullness over their respective sides only, and Grocco's sign is usually absent. There are, however, occasional exceptions to this rule. A case of aneurysm of the lower portion of the thoracic aorta, seen at the Philadelphia General Hospital, gave a typical Grocco's sign At post-mortem, a double-sac aneurysm was found, one sac on either side of the spinal column.

II. Relative or Moderate Dullness: *Normally,* relative dullness is elicited over those portions of the chest where the lung covers a solid organ; for example, in the third interspace to the left of the sternum, where the lung covers the heart and in the fifth interspace on the right side, where the lung covers the liver.

Pathologically, relative dullness is elicited over such morbid states of the lung and pleura as cause an admixture of a greater proportion of solid than air-containing structure Relative dullness may be elicited under the following conditions:

*1. **Intrapulmonary:** Small Consolidations* (bronchopneumonia, small tuberculous lesions) When percussing over a small consolidation we elicit not only the dull note characteristic of such tissue, but we also set into vibration the vesicular tissue immediately surrounding such a consolidation These vesicles usually enlarge because they compensate for the neighboring solid vesicles which have been put "out of commission" In consequence, we get an admixture of sounds, dullness from the consolidation, and res-

onance from the neighboring structures. This admixture can be best described as *dullness having some resonant quality,* properly named, *relative or moderate dullness.* This note is also elicited over deep-seated consolidations, deep-seated solid tumors, small infarcts and small areas of atelectasis. Edema of the lungs, fibroid phthisis and interstitial pneumonia likewise yield the same percussion note. The reason for relative dullness in these conditions is as follows:

Edema of the Lungs: In this condition we have in the air vesicles and their interstitial tissue an effusion of frothy, serous fluid, and under these conditions the proportion of airless substance (fluid) and air-containing tissue is such as to produce relative dullness. *Fibroid phthisis* and *interstitial pneumonia* have practically a similar admixture, *e. g.,* an overgrowth of fibrous tissue, followed later by shrinkage The partially shrunken air cells, which are well encased in airless fibrous tissue, so modify the percussion note that it yields relative dullness

2. ***Extrapulmonary Causes:*** Relative dullness is also elicited over thickened pleura, and small pleural effusions, mediastinal tumors, aneurysm, greatly hypertrophied heart, pericardial effusion, localized empyema and enlarged thymus.

III. **Impaired Resonance or Slight Dullness:** Impaired resonance is obtainable over those pathological conditions of the lung and pleura where airless tissue only slightly encroaches upon the air-containing element, so that the air-containing tissue predominates.

Such conditions as small tuberculous infiltrations, very small consolidations, small hemorrhagic infarcts, enlarged glands or very small solid growths, small atelectatic areas, or accumulations of exudate within the bronchi, lend a heightened pitch and slight impairment to an otherwise almost clear normal note. The same is true of a slightly thickened pleura, or a very scant pleural exudate.

B. Abnormal Clearness

In the normal chest there are areas over which clearness may be elicited. A clearer or more resonant note than normal over such portions is an indication of some abnormal condition, either of the particular area of lung lying directly beneath the point of percussion, *i. e.,* chronic emphysema, cavity, bronchiectasis or pneumothorax, or because of pathologic conditions existing in an adjacent portion of the lung, causing compensatory emphysema Compensatory emphysema causes enlargement of the lung vesicles, which accommodate more air than do other vesicles not so affected This enlargement is caused by the extra amount of air they are obliged to hold in order to compensate for the lack of respiratory air in a consolidated or otherwise diseased portion of lung lying adjacent to them. Because these vesicles contain more air, they give rise to a more resonant percussion sound. Just as the degree of dullness depends upon the amount of airless tissue added to the normal lung substance, so the degree of resonance is influenced by the quantity of air added to normal lung substance and the degree of pulmonary tension.

The abnormally clear sound may vary from mere exaggerated resonance to loud tympany. The intermediate steps are arbitrarily divided into:

I. Exaggerated resonance.

II. Hyperresonance.

III. Vesiculotympany, or skodaic resonance.

IV. Tympany—open, closed and their modifications, *i e*, cavernous, amphoric, Wintrich's change of sound, Gerhard's change of sound, Friedreich's phenomena and Williams' tracheal tone.

V. Cracked-pot sound.

I. **Exaggerated Vesicular Resonance** (puerile resonance): This sound is simply an increase in all the normal qualities of the normal vesicular note. It has the characteristics of vesicular resonance and can be readily recognized as such, differing only in that it is a trifle clearer and of somewhat lower pitch.

This sound is elicited over lung substance which contains a little more than the normal amount of air, all other relations of the lung to the surrounding structures remaining the same. The presence of this note indicates compensatory emphysema of short duration, before the vesicular walls have lost their elasticity. Such conditions will be found in an upper lobe of the lung as a result of moderate consolidation or compression of the lower lobe or *vice versa;* and on one side when moderate consolidation has taken place in the opposite lung. Exaggerated resonance disappears when the morbid condition responsible for this change is remedied. Exaggerated resonance may be slight or moderate, depending upon the degree of temporary distention. In young children, the normal chest note is one of exaggerated vesicular resonance, because the child's chest wall is thin and resilient, and also because of the greater intravesicular tension at that age

An exaggerated vesicular note is often elicited by percussing over the chest of anemic emaciated persons. In such cases there is a diminished amount of fat and muscle (airless tissue), and the skin is stretched tightly over the ribs The combination of a thinner substance to modify the lung resonance, and the increased resiliency of the ribs, are responsible for this note.

II. **Hyperresonance:** This is heard as an abnormally clear and deep note, both of greater intensity and longer duration. It was described by Biermer as a "bandbox note." This occurs in conditions where the lung vesicles are overdistended with air, and the vesicular walls have lost their elasticity, thus causing decreased pulmonary tension Hyperresonance is found in chronic bilateral emphysema. In this disease, because the lung vesicles are constantly overfilled, their walls become stretched to such an extent as to cause them to lose their elasticity. As a result of this, minute flabby air bladders are produced.

Hyperresonance is also obtained over a small unilateral pneumothorax.

III. **Vesiculotympany** (skodaic resonance): This is a combination of vesicular and tympanic resonance. The height of the pitch depends upon the greater predominance of the tympanitic quality over the vesicular quality. Vesiculotympany is closely akin to hyperresonance, differing only in pitch, the former having a slightly higher pitch, and a somewhat more tympanitic element. Hyperresonance is obtained over conditions of great pulmonary relaxation, while vesiculotympany is elicited over a large accumulation of air in the vesicles, with a lesser degree of relaxation In the language of Flint, who thus described this note: "The resonance is increased in intensity, the quality, a combination of the vesicular with tympanitic, and the pitch high in proportion as the tympanitic quality predominates over the vesicular. The sign

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II. **Hyperresonance:** This is heard as an abnormally clear and deep note, both of greater intensity and longer duration. It was described by Biermer as a "bandbox note." This occurs in conditions where the lung vesicles are overdistended with air, and the vesicular walls have lost their elasticity, thus causing decreased pulmonary tension Hyperresonance is found in chronic bilateral emphysema. In this disease, because the lung vesicles are constantly overfilled, their walls become stretched to such an extent as to cause them to lose their elasticity. As a result of this, minute flabby air bladders are produced

Hyperresonance is also obtained over a small unilateral pneumothorax

III. **Vesiculotympany** (skodaic resonance): This is a combination of vesicular and tympanic resonance. The height of the pitch depends upon the greater predominance of the tympanitic quality over the vesicular quality. Vesiculotympany is closely akin to hyperresonance, differing only in pitch, the former having a slightly higher pitch, and a somewhat more tympanitic element Hyperresonance is obtained over conditions of great pulmonary relaxation, while vesiculotympany is elicited over a large accumulation of air in the vesicles, with a lesser degree of relaxation. In the language of Flint, who thus described this note: "The resonance is increased in intensity, the quality, a combination of the vesicular with tympanitic, and the pitch high in proportion as the tympanitic quality predominates over the vesicular. The sign

represents especially, one morbid physical condition, namely, an abnormal accumulation of air in consequence of dilatation of the air vesicles—that is, pulmonary or vesicular emphysema. The sign is also present in interstitial or interlobar emphysema" A vesiculotympanitic resonance is obtained when percussion of the lung is carried out above a pleural effusion Although the pressure of the liquid diminishes the volume of the lung, it increases its tension, therefore making possible the production of this note. The note is vesiculotympanitic above a collection of liquid when this is sufficient to fill a third, or even a half or two-thirds, of the intrathoracic space. This note is also obtained over an upper lobe when the lower lobe is consolidated, as in the second stage of lobar pneumonia, and over the lower lobe when the upper lobe is consolidated.

Vesiculotympany is also obtained by percussing the lung adjacent to a large pericardial effusion, to a greatly hypertrophied heart, or a large solid tumor. An abnormally high position of the diaphragm due to any cause, will also produce this note at the base of the lung, this being due, no doubt, to the existence of pressure. In the normal chest vesiculotympany is elicited by percussing the left hypochrondriac region. In this region the stomach encroaches upon the lung, thus adding a tympanitic note.

IV. **Tympany** (tympanitic resonance): This may be defined as a clear hollow drumlike note which is entirely devoid of vesicular quality It is never found over normal lung substance within the body, and its presence, therefore, indicates a distinctly pathological condition.

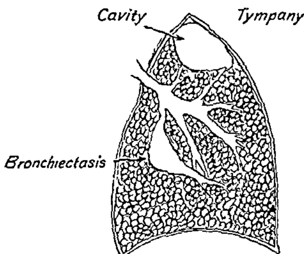


Fig 21—Cavity in lung yielding tympany.

The *pitch of tympanic sounds* depends upon the amount of relaxation of the lung structure, the amount of air in the cavity, and the size of its communicating opening with a bronchus. A lung that is very much relaxed produces a low pitch, as does also a very large lung cavity; while a small cavity with tense walls, small areas of relaxed lung, or a cavity communicating with a bronchus through a small opening, give rise to a high pitch.

Tympany can be elicited over: (*a*) A cavity in the lung filled with air; (*b*) a large bronchiectasis; (*c*) relaxed lung. (*d*) complete retraction of the lung; (*e*) occasionally during the first and third stages of lobar pneumonia; (*f*) pneumothorax (air in the pleura); (*g*) edema of the lung; (*h*) reflexly from the stomach; (*i*) peribronchial consolidations; (*j*) infarcts at the base of both lungs.

(*a*) ***Cavity:*** A localized area of tympany in the lung is usually caused either by a localized pneumothorax or by a pulmonary cavity A cavity in the lung may be due to destruction of the lung tissue by tuberculosis, gangrene, abscess or actinomycosis To differentiate by percussion between a pneumothorax and a cavity, one must bear in mind that a cavity is the result of a previous consolidation Therefore, when an area of dullness surrounds or is adjacent to the tympanitic area, it is an indication of the existence of a cavity, while in pneumothorax the note obtained over the area immediately surrounding the tympanitic area is exaggerated resonance The modified coin test will be found positive in the presence of a bronchiectatic cavity, but will be negative over a pulmonary cavity. The degree of tympany elicited over a cavity depends upon the following conditions:

(1) Size of the cavity
(2) Condition of its walls
(3) Its position
(4) The character of the tissue covering it.
(5) The character of its contents
(6) Whether it is directly or indirectly in communication with a bronchus
(7) The size of the communicating opening.

1. The larger the cavity, other things being equal, the more pronounced is the tympanitic note, and the lower is its pitch. The tympanitic notes elicited on percussion over the pathological chest are hardly ever as deep as those obtained over the stomach or the colon, because a lung cavity is much smaller than either of these organs; the tension of the lung and the composition of the chest wall are also, no doubt, in part responsible for the difference in pitch

A cavity in the lung, no matter how superficial, must be of a certain size before tympany can be elicited, if it is smaller than a pigeon's egg or a walnut, it will not yield a tympanitic quality.

2 A cavity with very lax walls will produce a low-pitched tympanitic sound which is spoken of as *cavernous.* A cavity having tense and resilient walls produces a high-pitched, almost metallic tympany which is called *amphoric.* Amphoric and cavernous resonance often merge in the same cavity

3 The *position* of a cavity has a marked influence upon the percussion note. The existence of a superficial cavity situated at the apex of the lung or elsewhere, even if no larger than a walnut, can be brought out by light percussion; a much larger cavity, when deep seated may be overlooked, even on forcible percussion The deeper the cavity, the more indistinct and muffled is the tympanitic quality. A cavity is easily demonstrated in an emaciated subject, particularly so if its location is superficial.

4 A superficial cavity covered with unaffected pleura usually produces a clear tympanitic sound. If the pleura is greatly thickened, dull or "wooden" tympany is elicited. The clearness of the tympanitic sound depends upon the amount of air-

represents especially, one morbid physical condition, namely, an abnormal accumulation of air in consequence of dilatation of the air vesicles—that is, pulmonary or vesicular emphysema. The sign is also present in interstitial or interlobar emphysema." A vesiculotympanitic resonance is obtained when percussion of the lung is carried out above a pleural effusion Although the pressure of the liquid diminishes the volume of the lung, it increases its tension, *therefore making possible the production of this note.* The note is vesiculotympanitic above a collection of liquid when this is sufficient to fill a third, or even a half or two-thirds, of the intrathoracic space. This note is also obtained over an upper lobe when the lower lobe is consolidated, as in the second stage of lobar pneumonia, and over the lower lobe when the upper lobe is consolidated.

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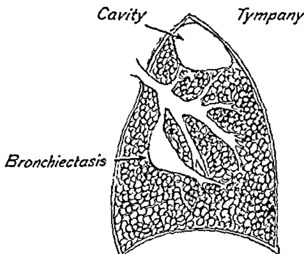


Fig 21—Cavity in lung yielding tympany.

IV. **Tympany** *(tympanitic resonance)* · This may be defined as a clear hollow drumlike note which is entirely devoid of vesicular quality. It is never *found over normal lung* substance within the body, and its presence, therefore, indicates a distinctly pathological condition.

The *pitch of tympanic sounds* depends upon the amount of relaxation of the lung structure, the amount of air in the cavity, and the size of its communicating opening with a bronchus. A lung that is very much relaxed produces a low pitch, as does also a very large lung cavity; while a small cavity with tense walls, small areas of relaxed lung, or a cavity communicating with a bronchus through a small opening, give rise to a high pitch.

the opening to be submerged under the fluid, thus converting it into a noncommunicating cavity. Readjusting the position of the patient will cause a return of the clearer tympanitic sound

4 *Gerhardt's Change of Sound:* This also consists of a variation in the percussion note accompanying a change of posture It may be elicited over a large cylindrical or oval cavity containing air and fluid, when the long diameter of the cavity corresponds to that of the lung, and of the body as a whole. With the patient in the sitting or standing position, a muffled low-pitched tympanitic sound can be elicited over the lower portion of the cavity, and a clear tympanitic sound above that; this is due to the gravitation of the fluid, so that the air occupies the upper part of the chamber. In the recumbent position the tympanitic note becomes clear over the anterior surface of the chest, owing to the uniform distribution of the air over the upper part of the cavity, while the fluid occupies the entire posterior surface of the cavity, and to the change of intracavity tension This sign cannot always be elicited; when present, it is an indication of a lung cavity. Gerhardt's change of sound may be obtained over a closed or an open cavity.

5. *Friedreich's Phenomenon·* This consists of an alteration of the cavity percussion note during respiration The tympanitic note is higher in pitch at the height of deep inspiration and is lower in pitch during expiration. The elevation in pitch during inspiration may be caused by the increased tension in the cavity and in the surrounding lung substance.

6. *Williams' Tracheal Tone* (rare): Normally, when percussing over the trachea, the tympanitic sound is louder and its pitch higher when the mouth is open, becoming lower in pitch when the mouth is closed. If the same change in sound can be elicited over the seat of the primary bronchus (first or second interspace near the sternum), it indicates retraction or consolidation of the apex. This sign should not be confounded with Wintrich's change of sound, which is found over a cavity and in addition presents all the other signs of a cavity, while the tracheal tone is obtained over retracted or infiltrated lung tissue only.



Fig 22—Technic for eliciting the coin test

7. *Biermer's Sign·* This consists of a change of pitch upon alteration of the patient's position It is elicited over a pneumothorax, or more likely, over a pyo- or hydropneumothorax. There must be an admixture of air and free fluid on the affected side, and it depends upon the same principles as does Gerhardt's change of sound. When the patient is in the recumbent position, the pitch is higher than when he is sitting or standing erect.

8 *Coin Test:* Whenever present this is a pathognomonic sign of pneumo-

less tissue covering it. The more airless tissue, the more muffled is the tympany.

5. The character of its contents markedly influences the tympanitic percussion sound. A cavity containing air produces a clear tympanitic note; if its contents be liquid and air, the note is altered in proportion to the amount of liquid it contains. A cavity filled entirely with secretion will yield a dull note; after the secretion has been expectorated, a tympanitic note can be obtained over the same area. A cavity half filled with liquid produces a note of less resonant quality and higher pitch, than does one without such encumbrance.

6. The note elicited over a cavity communicating directly with a bronchus differs markedly in pitch from that obtained over a cavity without direct communication. When there is no direct communication a low-pitched note is obtained which is termed *closed tympany.* If the cavity communicates directly with a bronchus it will give rise to a higher pitched and more drumlike sound, which is termed *open tympany.*

The pitch also varies in accordance with the size of the communicating opening; the larger the communication, the higher the pitch, and the more tympanitic the quality.

The pitch of the tympanitic sound elicited by percussing over a cavity may change under certain conditions:

1. *Wintrich's Change of Sound:* When percussing over a cavity communicating with a bronchus, the pitch is higher and the tone more tympanitic when the mouth is open, and will be lower when the mouth is shut. As the mouth is opened, the pulmonary cavity at once communicates with the outside air through this large orifice; therefore, the pitch is higher because "the larger the communicating opening the higher the pitch." When the mouth is shut, the cavity is transformed into a noncommunicating one, with resultant lower pitch.

Technic: The patient should stand or sit in an unconstrained position, facing the examiner. The examiner places his pleximeter finger firmly and evenly in the interspace over the site of the pulmonary cavity and directs the patient to open his mouth as if to pronounce the sound *Ah,* while the tongue is allowed to protrude slightly, and the breathing is superficial. The examiner strikes the pleximeter finger two or three light rapid strokes, and then directs the patient to close his mouth, and again strikes the same number of blows. This alternate opening and closing of the mouth may have to be performed several times before the change of sound is apparent to the examiner. The sound is much better elicited when the patient holds his open mouth near the examiner's ear.

2. The sound also varies with the position of the patient's lips; if the patient opens his mouth holding his lips in the position to pronounce *Ah,* the pitch is lower than if his lips are held in the position to pronounce *Ee.* Four notes of the scale can easily be elicited by having the patient change the position of his lips in the following order: *Oo—Ah—Ee,* then shutting the mouth. This range of sound variation can easily be elicited when percussing over the trachea of a normal subject. Wintrich's change of sound may also be elicited by percussing over an open pneumothorax, that is, one that communicates with a bronchus.

3. *Interrupted Wintrich's Change of Sound:* This sign is elicited when the communicating cavity contains fluid; changing the patient's posture may cause

the opening to be submerged under the fluid, thus converting it into a noncommunicating cavity. Readjusting the position of the patient will cause a return of the clearer tympanitic sound.

4. *Gerhardt's Change of Sound:* This also consists of a variation in the percussion note accompanying a change of posture. It may be elicited over a large cylindrical or oval cavity containing air and fluid, when the long diameter of the cavity corresponds to that of the lung, and of the body as a whole. With the patient in the sitting or standing position, a muffled low-pitched tympanitic sound can be elicited over the lower portion of the cavity, and a clear tympanitic sound above that; this is due to the gravitation of the fluid, so that the air occupies the upper part of the chamber In the recumbent position the tympanitic note becomes clear over the anterior surface of the chest, owing to the uniform distribution of the air over the upper part of the cavity, while the fluid occupies the entire posterior surface of the cavity, and to the change of intracavity tension. This sign cannot always be elicited; when present, it is an indication of a lung cavity Gerhardt's change of sound may be obtained over a closed or an open cavity.

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Fig 22—Technic for eliciting the coin test

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8 *Coin Test:* Whenever present this is a pathognomonic sign of pneumo-

thorax. This sign is obtained by placing a small coin (a 25- or 50-cent piece) on the chest, which an assistant holds in position and strikes with the edge of another coin, while the examiner auscultates the reverse side of the chest; that is, the coin is struck anteriorly as the



Fig 23—Author's modified coin test for determining the presence of a bronchiectatic cavity

examiner listens posteriorly, or *vice versa* A clear metallic, bell-like, ringing or chiming sound is heard (bell tympany) This sign should not be confused with the intensified note heard when a coin is struck while lying against a rib The sound thus produced is carried by the rib to its articulation, and can there be heard as a loud sound.

9. *Change of Line of Dullness on Change of Posture:* This is a sign pathognomonic of free fluid in any of the cavities of the body (pleural or abdominal); since water seeks its lowest level, the free fluid gravitates downward. One should guard against confusing this sign with a change of the upper line of liver dullness simulating pleural effusion. The presence of other physical signs will be of help in differentiating this particular condition.

Technic: Percussion should be started far above the seat of the effusion, while the patient stands or sits erect. The upper line of dullness is noted as it is approached from the clear or resonant part of the chest. The patient is then asked to recline toward the affected side, and percussion is again performed in the same manner. It will be noted that the upper level of dullness is much higher when the patient inclines toward the affected side He should now be asked to lean toward the sound side, whereupon percussion will demonstrate that the upper line of dullness is much lower than in either of the previous positions, the change being caused by the gravitation of the fluid.

10. *Bauti's Sign of Retrosternal Dullness:* This is observed in pleurisy with effusion, but is absent in pneumonia



Fig. 24—Author's modified coin test for determining the presence of a bronchiectatic cavity.

(*b*) ***Bronchiectasis:*** Bronchiectasis is a dilatation of a bronchus or of several bronchioles. It may be large or small, cylindrical or globular, single or

multiple, and may have the aspect of a pulmonary cavity which communicates directly with a bronchus. The percussion note varies according to the size of the dilatation, its position in the lungs, and the character of its contents. A sufficiently large bronchiectatic cavity, superficially situated, when free from secretion, will give rise to *high-pitched tympany* Small multiple bronchiectatic cavities, which communicate indirectly with one another, will give rise to *cracked-pot sound*

cumscribed empyema. It is also demonstrable at the bases of the lungs when they become relaxed because of pressure exerted upon them by an intraäbdominal neoplasm, or by an enlarged organ pushing the diaphragm upward.

(*d*) ***Completely Retracted Lung:*** In pulmonary retraction the bronchi may remain unencumbered; under these conditions the side of the chest in which this occurs assumes a drumlike resilience and readily imparts a low-pitched tympanitic sound.

Differential Diagnosis Between Bronchiectasis and Pulmonary Cavity

	Bronchiectasis	*Pulmonary Cavity*
	1 High-pitched tympanitic percussion note or cracked-pot sound	1. Tympany may be of high or low pitch, depending upon various conditions
	2 The area immediately surrounding the cavity yields a hyperresonant note	2. The area adjacent to, or immediately surrounding the cavity, yields a dull note
	3 The tympanitic note may give place to a dull note within a few hours, the tympanitic note returns when a large quantity of secretion is expectorated	3 The tympanitic note is usually constant
The Author's sign.	4 When a coin is placed upon the area overlying a bronchiectasis and is struck with another coin, a very much intensified note is heard by the examiner, as he places his ear or the stethoscope near the patient's open mouth	4. This sign, as a rule, cannot be elicited
	5. Usually situated posteriorly, near the base of the lung (when not of tubercular origin)	5. Usually situated in the upper part of the lung

(*c*) ***Relaxed Lung*** (pulmonary relaxation): The note elicited over a relaxed lung is a high-pitched tympany, with the peculiar "bandbox" quality termed skodaic resonance Such a note may be elicited in the normal chest by percussing Traube's semilunar space Pathologically, it may be elicited by percussing over relaxed lung tissue adjacent to a solid tumor, a greatly enlarged heart, a pericardial effusion, or a cir-

(*e*) ***First and Third Stages of Lobar Pneumonia:*** At times a tympanitic note is elicited over a portion of the lung underlying a consolidation, as at the beginning of the first stage of lobar pneumonia, and also during the later period of resolution in the third stage of pneumonia. This phenomenon may be explained in two ways: First, during the two stages of pneumonia just mentioned, the lung substance presents

thorax. This sign is obtained by placing a small coin (a 25- or 50-cent piece) on the chest, which an assistant holds in position and strikes with the edge of another coin, while the examiner auscultates the reverse side of the chest; that is, the coin is struck anteriorly as the



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Fig. 24—Author's modified coin test for determining the presence of a bronchiectatic cavity.

(*b*) ***Bronchiectasis:*** Bronchiectasis is a dilatation of a bronchus or of several bronchioles. It may be large or small, cylindrical or globular, single or

by the heavy percussion stroke forcing a large volume of air through a small opening. This sign is best elicited over a thin chest wall and during respiration Keeping the mouth open enhances its distinctness This sound may often be elicited over the chests of healthy children when crying.

Pathologically, it may be elicited over:

(*a*) Large *superficial lung cavities* free from fluid and communicating directly with a bronchus through a small opening.

(*b*) Pneumothorax communicating with a bronchus, especially when circumscribed.

(*c*) Pneumonic consolidations, particularly during the first and third stages in the upper lobes.

(*d*) Retracted lung tissue, especially above a large pleural effusion.

(*e*) Thoracic fistula, whether made by the surgeon or of accidental origin.

(*f*) Multiple bronchiectatic cavities which communicate indirectly with one another. This phenomenon has little significance when it occurs alone, because of its presence in such varying conditions, but is of importance as a corroborative sign

Lung Reflex

The lung reflex consists of a temporary localized emphysema; it occurs when a strong irritant has been applied to the chest, or as the result of the irritation produced by excessive and forcible percussion Hyperresonance is elicited during the stage of emphysema. This fact should be borne in mind when percussing over a suspicious area. Prolonged or too forcible percussion may change an impaired resonant note to hyperresonance

Lung Boundaries

The lung boundaries may vary to a greater or less extent, as a result of disease of the lung or its underlying structures. Bilateral extension of the lung boundary is found in chronic emphysema. Bilateral *diminution* of the lung boundaries is often observed in those who do not breathe deeply, in phthisical individuals, and in conditions which cause upward displacement of the diaphragm. Unilateral extension of the lung may be observed in compensatory emphysema; an inflated stomach or a pneumothorax may simulate this condition Unilateral retraction of the lung boundaries may be caused by shrinkage due to old pleural adhesions or to fibroid phthisis An enlarged liver, spleen or heart, or effusions in the pleural and pericardial sacs will sometimes simulate retracted lung borders, because of the encroachment of a dense tissue upon the resonant lung structure.

an admixture of consolidated and relaxed vesicles, the latter predominating. Second, small consolidated areas may be adjacent to a bronchus; the latter note is readily transmitted through the small consolidation.

(*f*) ***Pneumothorax:*** During the early stages of pneumothorax when the air pressure is not excessive, and before the appearance of a liquid effusion, a very loud amphoric or metallic tympany can be elicited over the affected side The lung above the pneumothorax gives a hyperresonant note, partly because of some relaxation and compression, and partly because the accumulated air in the pleura acts as a resonating chamber After the appearance of a liquid effusion (serum or pus), when it is of sufficient volume to exert pressure upon the pneumothorax, the amphoric tympanitic note changes to dull "wooden" tympany. This change is explained by the fact that the great intrathoracic pressure thus exerted deadens the vibrations, and the fluid within the pleura acts as a buffer and dulls the note, also, when the stage of pyo- or hydropneumothorax is reached, there is sufficient inflammatory exudate deposited upon the pleurae to cause them to become thickened and inelastic. *The presence of fluid in a pneumothorax* is manifested by a dull note over the fluid and a tympanitic note above its level. A change from the erect to the recumbent posture will cause a change in the level of dullness. There may also be present a succussion splash and metallic tinkling (SEE: p 347).

(*g*) ***Edema of the Lungs:*** If there is slight edema and the lung becomes relaxed in consequence, a tympanitic percussion note will be elicited.

(*h*) ***Reflex from the Stomach:*** A greatly distended stomach, pressing upon the diaphragm, may cause a tympanitic note at the base of the left lung Also, when a basal consolidation rests against an inflated stomach or colon, the tympanitic note common to the inflated organs may be transmitted upward so that it passes through the consolidated lung.

(*i*) ***Peribronchial consolidations,*** enlarged peribronchial glands or small neoplasms, when situated between the trachea and a bronchus, or between a large bronchus and the inner surface of the chest, may give rise to tympany on percussion, because the normal tympanitic note of these tubes is transmitted through the encroaching solid bodies as a dull high-pitched tympany.

(*j*) ***Infarcts at both bases of the lungs*** may cause tympanitic resonance when the infarcts are adjacent to a relaxed lung, or when the lung is in contact with a bronchus.

A sense of resistance, or a nonyielding sensation, is perceived by the pleximeter finger when a solid substance is percussed. A similar sense of resistance may be perceived by the plexor finger, which is imparted to the rounded edge of that finger, as a sense of dull pain The resistance perceived while percussing an airless tissue bears a direct relation to dullness Tissues producing the dullest sounds have the greatest resistance, and *vice versa,* tissues over which the clearest sounds are elicited impart the least sense of resistance.

V. Cracked-Pot Sound: This is a sound resembling, as its name indicates, the sound elicited by smartly tapping a cracked earthen pot, or a china vase; this sound may also be imitated by holding the palms of the hands lightly together and then striking them upon the knee. Cracked-pot sound is produced

ent sounds from different regions when heard at the same time.

L. Napoleon Boston greatly modified the Scott-Allison differential stethoscope so that it is of practical value in the comparative study of different regions.

Allison's hydrophone consists of a small rubber bag the size of a watch, which is filled with water. Allison claimed that sound is better conducted from the chest wall if water is used as an interposing medium. A binaural hydrophone was devised by D. M. Gamman consisting of G. P. Camman's binaural instrument with a water chamber above the chest piece.

S. Solis-Cohen's intrathoracic stethoscope consists of a rubber tube attached to the binaural stethoscope with a diaphragm of gold-beaters' skin, guttapercha or celluloid in a rubber capsule intervening between the two tubes. The capsule is passed down the esophagus

Bowles' stethoscope is a binaural instrument to which is attached the chest piece devised by Bowles. It is a shallow steel cap, the chest surface of which is fitted with a circular hard rubber or celluloid disk. This stethoscope is very practical, the smaller chest piece being adjustable to any surface. When pressed tightly against the chest wall, its flat surface will cause very little discomfort. Whenever necessary, it can be slipped under the back or side of a very sick patient who cannot be moved; it may also be slipped under a garment worn by the patient, if for any reason exposure is inadvisable. The sounds are brought out more clearly with this stethoscope, chiefly because of the shallowness of its air chamber, the diaphragm protecting the mouth of the chest piece having very little effect upon the transmission of sound. The instrument will transmit sounds just as readily when devoid of any diaphragm.

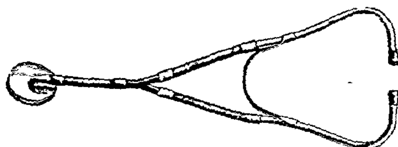


Fig 5—Binaural stethoscope, Bowles' chest piece.

The Gordon Stethoscope: Burgess Gordon modified the Bowles' chest piece. The cup is deeper and the edges are covered by a rubber cushion which does not require the use of a protective diaphragm. It is a very good instrument

Ford's stethoscope is a binaural instrument; the chest piece is rather long and in the essentials it differs little from any other binaural stethoscope It is the instrument preferred by the Army and Navy Medical Corps.

The *phonendoscope,* invented by Buzzi and Branchi, is a stethoscope which intensifies auscultatory sounds and also lends them a peculiar metallic tone. To one accustomed to auscultate with this instrument, the mental impressions of the various sounds will be such that he will often utterly fail to recognize the same sounds when the unaided ear is

taking pulse pressure, and at times also for locating fetal heart sounds.

The first authentic description of this instrument and a report of auscultatory findings as a result of its use, was given in 1819 by Laennec, a French clinician, in a publication entitled *Traité de l'Auscultation Mediate et des Malades des Poumons et du Coeur* (Mediate Auscultation and Diseases of the Lungs and Heart). Laennec first devised his stethoscope in 1816 It consisted of a cylinder of paper tightly rolled and held in shape by paste. From that time until the present, numerous instruments for auscultation have been devised, each being a trifle more serviceable or practical than its predecessor. There are now a number of very good stethoscopes on the market.

The *monaural stethoscope,* after its invention by Laennec, underwent successive modifications until the general adoption of Hawksley's instrument, which is still much used on the European continent and by some of the older clinicians in this country.

The *binaural stethoscope* is now the universal auscultation instrument in this country. Landouzy and March, in 1850, were among the first to construct the prototype of the instruments in use at present. G. P. Camman's binaural stethoscope, evolved in 1852, was the first model after which most modern stethoscopes are fashioned, each, however, having some modification of its own, which justifies its presence upon the market.

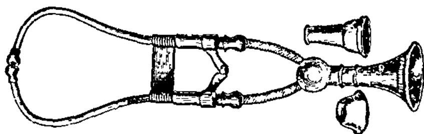


Fig. 3—Camman's stethoscope.

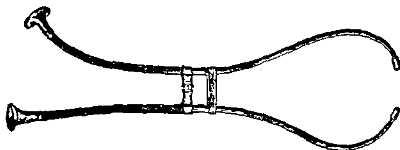


Fig. 4—Allison's differential stethoscope.

Scott Allison's differential stethoscope consists of two flexible monaural stethoscopes joined together by a spring, with two earpieces and two chest pieces This enables the examiner to listen to two different portions of the chest at the same time. The only practical application of this instrument is for simultaneous study of the diaphragmatic excursions of both sides It requires a good deal of practice to dissociate the ears sufficiently to be able to recognize differ-

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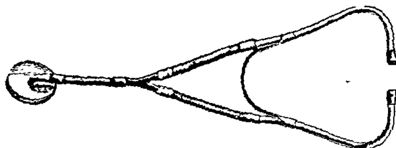


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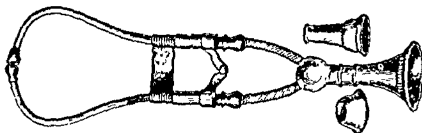


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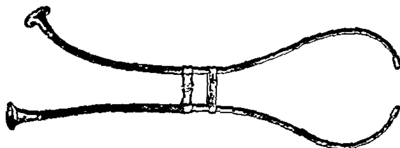


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obtained by listening to the left axillary or infraclavicular region Systematic auscultation should then be begun at the apices, the patient being allowed to breathe naturally It will prove less embarrassing, particularly with women, if auscultation is commenced posteriorly in the suprascapular region; by the time the posterior aspect of the chest has been thoroughly auscultated, any possible embarrassment will have subsided sufficiently to enable the examiner thoroughly and systematically to auscultate the anterior aspect

Auscultation of the lungs is performed in five successive steps:

1. **During Normal or Quiet Breathing:** The typical normal breath sounds are found in the left axillary and left infraclavicular regions of a normal person. In the infraclavicular regions the breath sounds are somewhat harsher than in the axillary regions. As in palpation and percussion, one region or intercostal space should be carefully compared to the corresponding region or intercostal space of the opposite side The examiner should listen in one spot to at least four or five respiratory cycles, before he attempts auscultation over another area Each intercostal space should be auscultated in no less than three vertical planes in each region of the chest. After the patient's chest has been thoroughly auscultated during quiet or normal breathing, the second step is begun

2 **During Deep Breathing** (preferably mouth breathing, the mouth being slightly open): The patient is instructed to breathe deeply but quietly, while the examiner repeats the examination with the same thoroughness as in step one.

3 **During Whisper:** The patient is asked to whisper one-two-three or any one of the stock phrases, and the distinctness of the transmitted whisper should again be noted in the various regions and intercostal spaces

4. **During Speech:** The patient is instructed to repeat in a loud voice such a stock phrase as *one-two-three*, or *ninety-nine*. The intensity of the voice transmission should be noted in each region and compared with the corresponding region on the opposite side.

5 **During Cough:** The final step consists in asking the patient to cough slightly after expiration, so that the influence of cough upon the respiration in the various regions can be noted. This procedure will often bring out râles previously inaudible, while at other times (depending upon the pathologic condition of the lungs and bronchi) coughing may cause râles to disappear or their location to change.

Breath Sounds

Three varieties of breath sounds are heard over the normal chest: 1. *Vesicular breathing, or normal lung sounds, normal vesicular murmur*—over normal vesicular lung structures; this sound being modified in the very young (puerile respiration) and in the very old (senile respiration); 2. *Bronchovesicular breathing*—where the smaller bronchi and lung substance meet, *i. e.*, second intercostal space near the sternum and the supraspinous fossae close to the spine; 3 *Bronchial breathing*—over a tubular structure, *i e*, the trachea and large bronchi.

These normal lung sounds may be classified as follows:

1. Vesicular Breathing

Quality: Vesicular or breezy.
Intensity · Soft or feeble.
Pitch: Low.

used, and for this reason its employment is rather to be discouraged.

In choosing a stethoscope the most essential requirement is *properly fitting earpieces*. It does not matter much what kind of chest piece is selected, provided it is not more than seven-eighths of an inch in diameter. In a short time one can accustom himself to any of the mod-

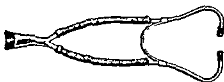


Fig 6—Binaural stethoscope, Ford's chest piece

ern chest pieces, but auscultation with any instrument, the earpieces of which do not fit properly, will be found worse than useless The external auditory meatus is not of the same size in all persons, therefore, one must select earpieces which fit his individual ears. The earpiece should not be small enough to enter the auditory canal to any depth, but should be sufficiently large to cover the meatus completely

Caution: After using a certain size of earpiece for a number of years, one often finds it necessary to get a larger size, as the external auditory canal stretches from the prolonged use of the stethoscope

The *metal tubes* to which the earpieces are attached, should be curved slightly forward and downward to conform to the general direction of the auditory canal.

The *spring* which holds the metal tubes in position should not be too stiff. A very stiff spring will cause pressure pain to the ears. It should exert just enough pressure to hold the earpieces in position.

The *rubber tubing* should be fairly thick and of a length of about 12 to 14 inches. The inside diameter need not be very large, but the tube should be elastic in order to facilitate movements of the head in any desired direction.

Any of the popular *chest pieces* will serve the purpose of the clinician Each physician becomes accustomed to his own instrument, and cannot hear as well with another's even though it be the best stethoscope made. The chest piece should be of small circumference, should not be applied to the patient's body when cold, and should always be held by as few of the examiner's fingers as will enable him to grasp it firmly.

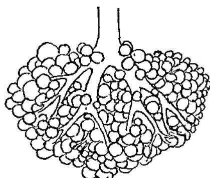
In speaking of Laennec and the instrument which he placed in the hands of the medical profession, C T Williams,[1] a well-known English thoracic specialist said· "No method, however, is so simple as that of auscultation, and the stethoscope remains an instrument which all medical practitioners ought to know well, for good hearing and patience is all that is required Some patients have no sputum to test and the shadows of the x-rays may be capable of many explanations Auscultation, therefore, holds its own, and will continue to do so to the end of time."

Technic

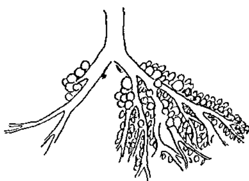
In auscultation, as in the other methods of examination, the position of the patient and of the physician must be easy and unconstrained. The patient must bare his chest and should be made to feel perfectly at ease.

In each case the standard normal vesicular breath sounds should first be

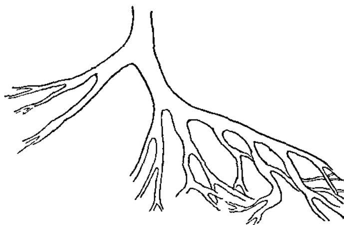
[1] Williams, C. T.: Laennec and the Evolution of the Stethoscope. British Medical Journal, July 6, 1907, vol ii, pp. 6-8.



1 All lung vesicles are filled during inspiration



2 Many of the lung vesicles remain airless during inspiration



3 None of the vesicles contain air.

Fig 7—Breathings. 1, Vesicular; 2, bronchovesicular; 3, bronchial.

Duration: Inspiration longer than expiration.
Rhythm: Inspiration and expiration occur regularly, and at a given number of times per minute.

2. Bronchovesicular Breathing

Quality: *Somewhat muffled, blowing*
Intensity: Somewhat harsh.
Pitch: Higher than vesicular, not quite so high as bronchial.
Duration: Inspiration two-thirds as long as expiration.
Rhythm: Regular.

3. Bronchial Breathing

Quality: Blowing, piping, tubular.
Intensity: Harsh.
Pitch: High.
Duration: Inspiration as long as expiration
Rhythm: Regular.

Normal Vesicular Breathing: It is evident from what has been said that the quality of the breath sound depends largely upon the structure of the tissue modifying it. Its analogy is found in wind instruments where the variations are often due to the difference in the caliber of the reed. The inspiratory sound begins in the larynx and is modified as it descends to the bronchi, bronchioles and vesicles.

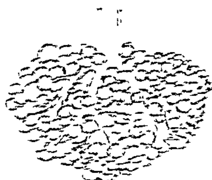
Every *respiratory sound consists of* two distinct parts, *inspiration* and *expiration,* which are separated from each other by a pause. It is important to note the quality of the breath sounds and the length of the inspiratory and expiratory sounds, their proportion to each other, and the length of the intervening pause.

Inspiration: *It should be emphasized* at the outset that the length of the inspiratory *act* bears no relation to the length of the inspiratory *sound* as heard over normal vesicular lung structure. The inspiratory *act* is shorter than the expiratory *act,* but the inspiratory *sound,* as heard over that portion of the chest overlying normal vesicular lung structure, is longer than the expiratory *sound;* thus, clinically speaking, it is stated, *when referring to vesicular sounds, that inspiration is three times longer than expiration.* The proportionate lengths of the inspiratory and expiratory *acts* can readily be noted by listening with the stethoscope over the mouth and nose of a sleeping person. It will be noted that the proportionate length of inspiration to expiration is reversed when listening over the mouth and nose of a sleeping person to that obtained by listening over the chest.

The inspiratory sound of normal vesicular *breathing commences as soon as* the air begins to enter the vesicular structures and lasts until they are entirely filled. The sound thus produced may be somewhat simulated by holding the lips in the position required to pronounce the sound F, at the same time taking a long breath. The ratio of the inspiratory sound *to the expiratory* is about three to one; the former is also a little harsher and louder than the latter.

Expiration: The expiratory sound of *normal vesicular breathing* as heard over the chest is the shortest breath sound encountered Any pathologic variation of the expiratory sound *will always* be a lengthening, because it is impossible for it to be shorter than the normal. This sound may be imitated by holding the lips in position to pronounce the letter V, and at the same time exhaling quickly; the sound will be soft and of low pitch; a mere whiff, often scarcely audible. The expiratory *sound depends* upon the collapse of the vesicular lung structure.

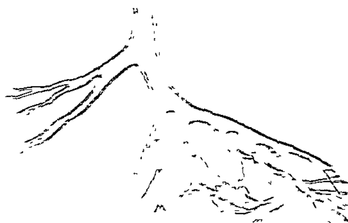
The difference in the length of both sounds may be explained by noting that



1. All lung vesicles are filled during inspiration.



2. Many of the lung vesicles remain airless during inspiration.



3. None of the vesicles contain air.

Fig. [illegible]—Breathing. 1, Vesicular. 2, Bronchovesicular. 3, Bronchial.

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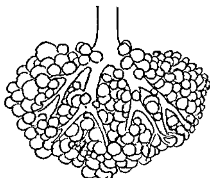
Every respiratory sound consists of two distinct parts, *inspiration* and *expiration,* which are separated from each other by a pause It is important to note the quality of the breath sounds and the length of the inspiratory and expiratory sounds, their proportion to each other, and the length of the intervening pause.

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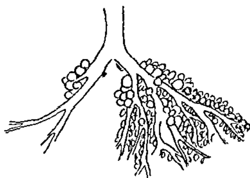
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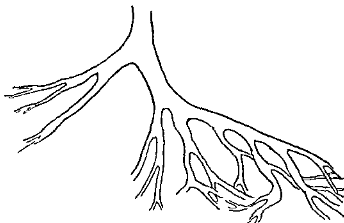
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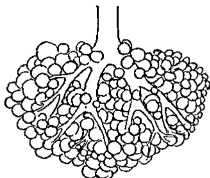
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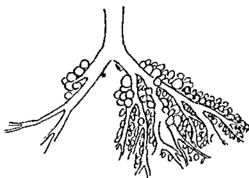
The inspiratory sound of normal vesicular breathing commences as soon as the air begins to enter the vesicular structures and lasts until they are entirely filled. The sound thus produced may be somewhat simulated by holding the lips in the position required to pronounce the sound F, at the same time taking a long breath. The ratio of the inspiratory sound to the expiratory is about three to one; the former is also a little harsher and louder than the latter.

Expiration: The expiratory sound of normal vesicular breathing as heard over the chest is the shortest breath sound encountered. Any pathologic variation of the expiratory sound will always be a lengthening, because it is impossible for it to be shorter than the normal. This sound may be imitated by holding the lips in position to pronounce the letter V, and at the same time exhaling quickly; the sound will be soft and of low pitch; a mere whiff, often scarcely audible. The expiratory sound depends upon the collapse of the vesicular lung structure.

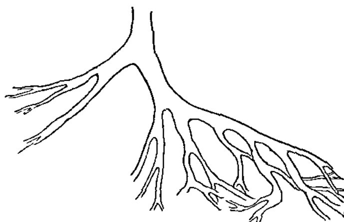
The difference in the length of both sounds may be explained by noting that



1 All lung vesicles are filled during inspiration



2. Many of the lung vesicles remain airless during inspiration



3 None of the vesicles contain air

Fig. 7—Breathings: 1, Vesicular; 2, bronchovesicular; 3, bronchial

Duration: Inspiration longer than expiration.
Rhythm Inspiration and expiration occur regularly, and at a given number of times per minute.

2 Bronchovesicular Breathing

Quality: Somewhat muffled, blowing
Intensity: Somewhat harsh
Pitch: Higher than vesicular, not quite so high as bronchial.
Duration: Inspiration two-thirds as long as expiration.
Rhythm: Regular.

3 Bronchial Breathing

Quality: Blowing, piping, tubular.
Intensity: Harsh.
Pitch: High.
Duration: Inspiration as long as expiration
Rhythm: Regular.

Normal Vesicular Breathing: It is evident from what has been said that the quality of the breath sound depends largely upon the structure of the tissue modifying it. Its analogy is found in wind instruments where the variations are often due to the difference in the caliber of the reed. The inspiratory sound begins in the larynx and is modified as it descends to the bronchi, bronchioles and vesicles.

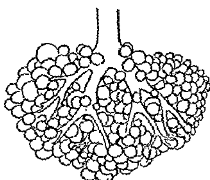
Every respiratory sound consists of two distinct parts, *inspiration* and *expiration*, which are separated from each other by a pause. It is important to note the quality of the breath sounds and the length of the inspiratory and expiratory sounds, their proportion to each other, and the length of the intervening pause.

Inspiration: It should be emphasized at the outset that the length of the inspiratory *act* bears no relation to the length of the inspiratory *sound* as heard over normal vesicular lung structure. The inspiratory *act* is shorter than the expiratory *act*, but the inspiratory *sound*, as heard over that portion of the chest overlying normal vesicular lung structure, is longer than the expiratory *sound;* thus, clinically speaking, it is stated, when referring to vesicular sounds, that *inspiration is three times longer than expiration.* The proportionate lengths of the inspiratory and expiratory *acts* can readily be noted by listening with the stethoscope over the mouth and nose of a sleeping person. It will be noted that the proportionate length of inspiration to expiration is reversed when listening over the mouth and nose of a sleeping person to that obtained by listening over the chest.

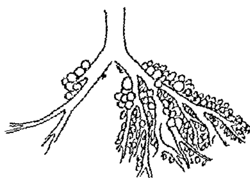
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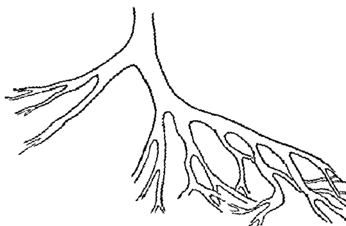
The difference in the length of both sounds may be explained by noting that



1 All lung vesicles are filled during inspiration



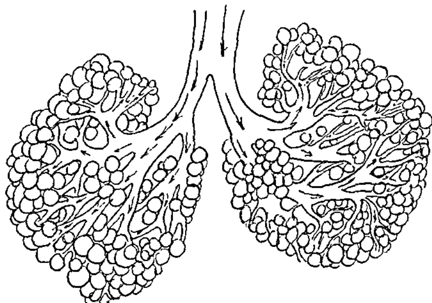
2 Many of the lung vesicles remain airless during inspiration.



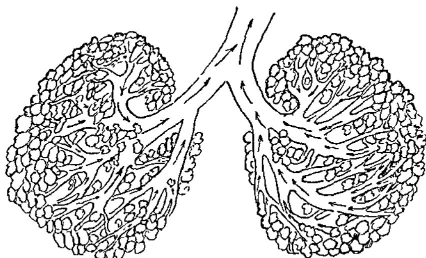
3 None of the vesicles contain air

Fig 7—Breathings · 1, Vesicular, 2, bronchovesicular; 3, bronchial.

it takes longer to fill a vessel through a small opening than it does to *empty* a similar vessel through a large opening. is only heard at the time the vesicles collapse *simultaneously;* when the air reaches the larger caliber tubes the



1 Inspiration. Air is being forced into the lungs against resistance from large tubes to successively smaller tubes until the vesicles are reached



2. Expiration The air is forced out of the lungs by the collapse of the air vesicles; it then passes through ever-larger nonresisting bronchi

Fig. 8—1, Inspiration. 2, Expiration.

During inspiration, the air has to pass through larynx, trachea, bronchi and bronchioles to the air vesicles and always against resistance. The expiratory sound sound is lost because of lack of resistance; it oozes out through the larger tubes. However, if, during expiration, the stethoscope is *held over the nose or*

mouth, the expiratory sound will be audible much longer than the inspiratory sound.

The normal vesicular *murmur* (inspiration and expiration) is spoken of as "soft" and "breezy," resembling the sound produced by a gentle wind rustling the leaves in a tree. The *pause* between inspiration and expiration is very short, often not at all perceptible. As soon as inspiration is completed expiration begins. A lengthening of the pause is an indication of some pathological condition.

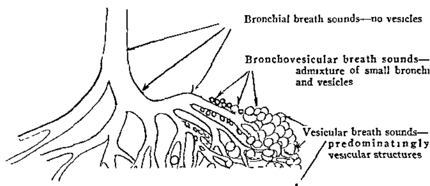


Fig. 9—Breath sounds heard over various parts of the normal chest.

Normal Variations

Normally, the vesicular murmur varies with age and sex, as for example

Puerile respiration is heard in children under 12 years of age where because of the resiliency of the chest wall and the elasticity of the vesicular structure both inspiration and expiration are much harsher, louder and longer, though still of vesicular quality. The inspiratory and expiratory sounds are also proportionately lengthened. However, the ratio between inspiration and expiration is maintained Thus we have:

NORMAL VESICULAR BREATHING
Inspiration, 3
Expiration, 1

PUERILE RESPIRATION
Inspiration, 6
Expiration, 2

In *senile respiration* the intensity of the vesicular murmur is diminished and indistinctly transmissible due to the weakened and inelastic condition of the lung. Expiration is somewhat prolonged and the intrarespiratory pause is somewhat lengthened.

Respiration is louder in *females* than in *males*, particularly in the upper part of the chest.

Muscular persons with thick chest walls present a feeble respiratory murmur because the added thickness interferes with the transmission of sound; on the other hand, those having *thin chest* walls transmit the vesicular mur mur more clearly.

Persons of sedentary habits and those who do not breathe deeply, present a

weak vesicular murmur because of insufficient development of the lungs. This is most noticeable at the borders of the lungs, that is, the apices and bases.

Bronchial Breathing (normal): Bronchial breathing is a harsh tubular piping sound. Inspiration is as long as expiration, both having the same harsh quality. It may be approximately imitated by sounding the German *ch*. The ratio between inspiration and expiration is three to three, and the interrespiratory pause is lengthened. Normally, bronchial breathing is heard anteriorly over the trachea and posteriorly over the spine of the seventh cervical vertebra; also over the skull particularly over the temporal regions.

Bronchovesicular Breathing (normal): *This type of breathing*, as its name indicates, is a combination of bronchial and vesicular; to be more exact, it is not as harsh as bronchial, but harsher than vesicular. Inspiration is a little harsher than the normal vesicular inspiratory sound, yet it retains a distinct *vesicular quality tinged with a* bronchial element. Expiration is a little longer, more intense and of higher pitch than vesicular breathing, having quite a bronchial element. The ratio between inspiration and expiration is as three to two. The interrespiratory pause is somewhat longer than that of vesicular breathing, but shorter than in the bronchial type.

Inspiration	Inter respiratory pause	Expiration
Normal Vesccular 3		1
Broncho Vesccular 3		2
Bronchial 3		3
Emphysematous 3		3½
Puerile and Compensatory Emphysema 6		2

Fig. 10—Inspiratory, expiratory ratio of the various types of breathing.

Bronchovesicular breathing is not as distinct a type as either bronchial or vesicular; there are many variations ranging from very mild to harsh breathing; its distinctive quality, however, is an intermediate position between bronchial and vesicular.

Normally, this type of breathing is heard where there is a blending of bronchi and vesicles as:

1. In the right second interspace, close to the sternum.

2. At the vertebral borders of the interscapular regions, and at the root of the lungs.

The harsher respiratory sound heard over the right supra- and infraclavicular regions can hardly be classed as typically bronchovesicular. It is simply a harsher vesicular murmur due to the anatomic peculiarities of the right bronchus.

Résumé:

	Inspiration		Expiration
Bronchial breathing	Harsh.	Intensity	Harsh.
	Long	Duration	Long.
	Tubular.	Quality	Tubular.
	Inspiratory-expiratory.	Ratio 3 3.	
Vesicular breathing	Soft	Intensity	A mere puff
	Longer.	Duration	Very short
	Breezy.	Quality	Very soft.
	Inspiratory-expiratory:	Ratio 3:1	
Bronchovesicular breathing	Soft, though tempered	Intensity	Fairly harsh
	Fairly long.	Duration	Not quite so long
	Mixed	Quality	Somewhat harsh
	Inspiratory-expiratory·	Ratio 3 2.	

Regional Auscultation

The breath sounds vary greatly in the different regions of the healthy chest owing to:

1. The variations of the lung structure.

2. The peculiar distribution of the bronchi.

3 The encroachment of other organs upon the lung

4. The variations in the thickness of the chest wall.

It is, therefore, necessary for the student to familiarize himself with the breath *sounds normally heard in the various re*gions or intercostal spaces, so that he may readily recognize the occurrence of the abnormal

Supraclavicular Regions:

Left

The breath sounds are vesicular but somewhat distant, because the lung apex has less volume than the other parts of the lung, and is further removed from the surface This region and the corresponding region on the opposite side should be carefully auscultated, as it is often the primary seat of manifest pulmonary tuberculosis.

Right

The breath sounds are somewhat harsher than on the left side, and expiration is prolonged.

Infraclavicular Regions:

Left

Auscultation should properly begin at this region, because typical normal vesicular breath sounds are here heard, and it can be taken as a standard for each individual

Normal vesicular breathing is heard in the first and second interspaces, in the second interspace half an inch from the sternum Close to the sternum at this level, we can hear distinct bronchovesicular breathing, because of the entrance of the left bronchus in the lung. At the lowermost portion of the second interspace and over the third rib, we get exaggerated or puerile breath sound, caused by slight compression of the lung by the base of the heart.

Right

The breath sounds in this region are vesicular and bronchovesicular The vesicular breathing is much harsher than on the left side, expiration being prolonged because the right bronchus is *more direct, shorter, and of larger caliber* In the second interspace, near the sternum, distinct bronchovesicular breathing is heard, because at this level the right bronchus enters the lung superficially, and also because of slight compression of the lung by the base of the heart, and by a portion of the aortic arch

Mammary Regions:

Left

Third interspace in lean persons, slightly exaggerated breathing because the lung is somewhat compressed by the heart Fourth to sixth ribs, inside the parasternal line, distant breath sounds are heard because of the position of the heart. In that region, outside the parasternal line, distant vesicular breathing is heard

Right

Third rib to fourth interspace somewhat distant, but pure vesicular breath sounds Fourth to fifth interspace somewhat exaggerated vesicular breathing, because the lung is buoyed up by the liver; below that level no breath sounds are heard because of the position of the liver.

Inframammary Regions:

Left

No breath sounds can be heard during quiet breathing because the lung rarely dips into this region, during deep breathing vesicular breath sounds are audible.

Right

No breath sounds during quiet breathing because this space is occupied by the liver

Superior Sternal Regions: The breath sounds in the suprasternal notch and over the uppermost portion of the sternum are bronchial because of the position of the trachea. Below Louis' angle, and on either margin of the sternum, as far as the third rib, the breath sounds are bronchovesicular.

Inferior Sternal Regions: No breath sounds are audible on the left border of the sternum, because of the presence of the heart, which lies beneath Very faint breath sounds are heard at the right border.

Superior Axillary Regions (armpit to sixth rib): On the *right* side the respiratory vesicular murmur is somewhat harsher than on the left side because of the extra lobe and slight compression of the lung by the liver The breath sounds heard in the *left* superior axillary region are purely vesicular, and may act as a standard for the normal quality of the individual The vesicular murmur *is*, however, distinctly audible on both sides.

Inferior Axillary Regions (sixth rib downward): *Breath sounds are* audible only to the eighth rib because, lat-

erally, the lung does not extend below that level. The vesicular murmur is distant and feeble, though distinctly audible on both sides.

Supraspinous Fossa or Suprascapular Region:

LEFT	RIGHT
Harsh vesicular breathing near the spine; distant vesicular in the remaining portion.	Modified bronchovesicular breathing near the vertebral spine Harsh vesicular with slightly prolonged expiration in the remaining portion.

Scapular Regions: The breath sounds are very distant and at times inaudible, particularly in quiet breathing, or in stout individuals, as the breath sounds are lost in passing through the scapulae.

Interscapular Regions: Bronchovesicular breathing is heard near the vertebral spine on either side. Over a small area the size of a half dollar, situated one inch away from the vertebral spine on either side, and on a level with the fourth intercostal space (fifth dorsal spine) there is heard bronchial breathing. This *area corresponds to the roots of the* lungs Over the remaining portions of either interscapular region only distant vesicular breath sounds can be heard

Infrascapular Regions (seventh to tenth ribs). In these regions the breath sounds are distinctly vesicular, though somewhat distant. An examination of the lungs is not complete unless these regions have been thoroughly auscultated, because they are the preferred sites of lobar pneumonia, and — not infrequently — of pulmonary tuberculosis, pleurisy, bronchitis, syphilis and malignancy of the lungs

Spinal Column: Over the spine of the seventh cervical vertebra, cavernous breathing is heard; over the second and third dorsal vertebrae, distant bronchial *breathing; below that level the breath* sounds become more distant.

Head Auscultation: Over the temporal regions cavernous breathing and over the parietal region, bronchial breathing is heard

Pathologic Breath Sounds

The breath sounds which have been described are normal for each region noted; any variations therefrom must, therefore, be regarded as pathological.

Pathologic Variations of the Normal Vesicular Murmur (Breath Sounds)

The normal vesicular murmur is spoken of as having five attributes:

I. **Intensity:** Soft or feeble

II. **Rhythm:** Inspiration and expiration occur regularly, and at a given number of times per minute. Adult male, 18 to 20; adult female, 20 to 22; children at birth, 44; at five years old, 25.

III. **Pitch:** Low.

IV. **Duration:** Inspiration longer than expiration.

V **Quality:** Vesicular or breezy.

The vesicular lung structure may be so distorted by disease as to give rise to the following modifications of the normal vesicular murmur:

I. **Alterations in Intensity:** Intensity may be (*a*) increased, (*b*) diminished, or (*c*) absent

(*a*) ***Increased Vesicular Murmur:*** This is a greater degree of loudness of the normal vesicular breath sounds. The ratio of inspiration to expiration is maintained, though both are somewhat prolonged, as found in compensatory emphysema. It is usually an indication of increased functional activity as a result of disease in an adjacent portion of the same lung, or of the opposite lung It may also occur in any portion of the lung as a result of partial compression or slight relaxation

(*b*) ***Diminished Vesicular Murmur*** (shallow or extreme senile respiration): The vesicular breath sounds are feeble, inspiration is shortened and expiration is often inaudible. It may occur as a result of:

1. Defective transmission of breath sounds due to (*a*) thickened chest wall, *i. e.,* edema, tumor, hypertrophied muscle or fat; (*b*) thickened pleura, or (*c*) a slight amount of pleural effusion.

2. Defective lung expansion, resulting from (*a*) partial obstruction of the trachea or of a bronchus by a tumor or a foreign body, or by secretion or edema; (*b*) paralysis of the diaphragm or thoracic muscles; (*c*) willfully holding the breath, because of pain due to peritonitis, pleurodynia, or intercostal neuralgia; (*d*) upward enlargement of the spleen, liver or stomach, or a tumor which causes upward displacement of the diaphragm, which in turn prevents lung expansion.

3. Diminished elasticity of the lung vesicles, as in edema, congestion of the lungs and chronic emphysema in the aged or feeble.

(*c*) ***Absent or Inaudible Breath Sounds:*** This may be caused by: (*a*) Large pleural effusions of serum, pus or blood, coincidentally pushing the lung away, and acting as an intervening medium; (*b*) large diffuse pneumothorax; (*c*) greatly thickened pleura; (*d*) fibroid phthisis causing shrinkage of the lung; (*e*) atelectasis or collapse of the lung from any cause, accompanied by occlusion of the bronchus; (*f*) extensive tuberculous deposits affecting the lung and pleura, and plugging of the bronchus, and (*g*) foreign bodies completely plugging a bronchus.

II. **Alterations in Rhythm:** Normally, inspiration and expiration occur regularly at a constant rate; pathologically, rhythm may be affected by: (*a*) Increase in frequency; (*b*) decrease in frequency; (*c*) irregular frequency; (*d*) interrupted inspiration; (*e*) shortened inspiration; (*f*) prolonged expiration, and (*g*) lengthened interval between inspiration and expiration.

(*a*) ***Increased Respiratory Frequency:*** This may result from the following causes:

I. *Physiologic:* Running, jumping or other violent physical exertion, and mental or psychical disturbances

II. *Pathologic:* 1. *Diseases of the lungs:* The pneumonias, pneumonoconiosis, bronchiectasis, moderately advanced and advanced pulmonary tuberculosis; consolidation or compression of one lung or of a lobe; pulmonary edema; congestion; asthma; emphysema; partial obstruction to the entrance of air in the lungs; or any condition that will cause a diminished aerating surface Tumors, aneurysms, diseases of thorax, diaphragmatic abscess, hernia, evisceration, etc., will cause rapid breathing because of mechanical interference.

2. *Diseases of the Heart:* Dilatation of one or more of the heart chambers, particularly of the left ventricle; degeneration of the myocardium, or any other

condition that may interfere with the action of the heart and cause cardiac decompensation.

3 *Disease of the Kidneys:* By causing edema of the lungs and effusions in the pleura, pericardium and peritoneum, and also because of failure to eliminate some of the toxins

4. *Febrile Disease:* By causing more rapid oxidation of tissue, thus producing toxins and probably, also, by direct action upon the respiratory centers.

5 *Disease of the Blood* All forms of anemia because of an insufficiency of erythrocytes to carry on proper oxygenation of the blood and also because of the blood being too poor in quality to nourish properly the respiratory apparatus

6 *Drugs:* Excessive doses of strychnine, alcohol, belladonna and its derivatives, etc.

7 *Nervous Origin:* Irritation of the respiratory center by tumor, embolism, shock, hysteria and other nervous affections

(*b*) ***Decreased Respiratory Frequency:*** This may be caused by poisoning with opium or its derivatives; uremia, diabetic coma; and other types of coma, certain brain affections; shock; hysteria; stenosis of the larynx; chronic fibroid phthisis when the patient is at rest, or approaching dissolution

(*c*) ***Irregularity as to Frequency:*** This is noticed in the terminal stage of certain nervous affections and in *Cheyne-Stokes breathing,* a variety of irregularity associated with cerebral, renal, cardiac, and pulmonary affections, as a rule, occurring shortly before death. It consists of a definite cycle divided into three distinct periods At first the respirations are deep, regular and slow; then they gradually become faster and shallower until they are very rapid and superficial; this stage is followed by a third stage, a period of apnea or suspended respiration, after which the cycle commences anew.

Biot's respiration consists of rapid, short respirations, interrupted by short pauses, lasting a fraction of a minute. This is seen in meningitis and rarely, in healthy subjects, during sleep.

(*d*) ***Interrupted Inspiration:*** The inspiratory sound, instead of being low-pitched, continuous and even, may become higher in pitch, jerky. "cog-wheeled" or granular

In *jerky inspiration,* each inspiratory sound is interrupted by an irregular number of sudden stops and jerks.

Cogwheel inspiration is practically a form of jerky inspiration, except that the stops occur regularly, the inspiratory sound being interrupted by two, three or even four, distinct stops.

Granular inspiration is a subdivision of the previous type, varied only by the occurrence of more stops, sometimes from eight to ten in each inspiration, these inspirations are not very deep and are often difficult to perceive, and the breath sounds convey a sensation similar to that which one experiences when he draws his finger over a sandy board Interrupted inspirations are met with in:

1. The first stages of acute plastic pleurisy.
2. Pleurodynia.
3. Incipient pulmonary tuberculosis (over the lesion).
4. Imperfect expansion of some portion of the lung (apical and basal).
5. Interrupted inspiration may also be met with in healthy subjects during the first deep inspiratory effort, which may cause full expansion of a hitherto imperfectly expanded portion of the

lung; frequently met with in clerks or others of sedentary occupation. After several deep inspirations, the interruptions disappear

(*e*) *Shortened inspiration* This may occur as a result of imperfect lung expansion; bronchial and asthmatic breathing also present this phenomenon.

(*f*) *Prolonged Expiration.* It has been pointed out before that the expiratory sound of normal vesicular breathing is very short because of the sudden collapse of the elastic air vesicles; if the air vesicles lose their elasticity, they are unable to collapse suddenly, and only by slow contractions permit the air to ooze out gradually, thus producing a prolonged expiratory sound Any condition which will bring about such a state will also cause a fibrosis of the bronchioles, thus transmitting the expiratory sound with greater intensity. A similar prolongation of the expiratory sound occurs as a result of consolidation of the lungs, a condition in which the air vesicles have been put out of service, and respiration is being carried on entirely by the bronchi. The same volume of air entering and leaving the same set of tubes without being split up, will naturally consume an equal length of time in its exit as it does in its entrance. Prolonged expiration is among the earliest physical signs in manifest incipient tuberculosis; its presence denotes congestion

1. *Prolonged Expiration—in Emphysema* · Expiration is as long or longer than inspiration, it is of low pitch and feeble vesicular quality, and can be heard over the entire chest.

2. *Large consolidation* is indicated by bronchial breathing; expiration is as long as inspiration and is of high pitch and tubular quality; it is heard over a portion of the chest overlying a consolidated lung, an exposed bronchus, or over the trachea

3. *Small consolidation* produces bronchovesicular breathing; expiration is two-thirds as long as inspiration and is of a modified tubulovesicular quality and moderately high pitch

4. Prolonged expiratory sounds are heard over a *large cavity*, particularly if the cavity communicates directly with a bronchus through a small opening. The inspiratory sound is also prolonged.

(*g*) *Lengthened Interval Between Inspiration and Expiration* · Normally, the pause between inspiration and expiration is hardly perceptible. A lengthening of this pause may be due to shortened inspiration, causing a greater interval; or to delayed expiration, the expiratory sound being delayed because of inelasticity of the vesicular lung structure. This condition is seen in cases of chronic emphysema.

III. **Alteration in Pitch:** The pitch of the respiratory murmur depends upon the degree of elasticity in the respiratory tract Thus: The *normal vesicular murmur* is of low pitch; *emphysematous breathing,* because of loss of elasticity in the vesicular structures, produces a still lower pitch. Per contra, *compensatory emphysema,* which causes a greater elasticity of the vesicular structures, produces a much higher pitch than normal vesicular breathing. *Bronchial* and *bronchovesicular breathing,* because of the increased tension in the respiratory tract, have a still higher pitch, the pitch being higher in bronchial than in bronchovesicular breathing.

The pitch is higher in *amphoric* than in *cavernous* breathing, because a cavity with tense walls which causes the amphoric breath sounds is a better resonat-

ing chamber than a cavity with flaccid walls which is the cause of cavernous breath sounds. However, both amphoric and cavernous breath sounds are of a lower pitch than either bronchovesicular or bronchial breathing.

IV. **Duration:** By *duration* is meant the length of time the sound is heard Any condition that will cause increased resonance will also lengthen its duration.

V. **Alteration in Quality:** The *quality* of the breath sounds depends upon their origin The breath sounds produced by normal vesicular lung structure have a breezy or vesicular quality. If the air vesicles are under tension they produce sounds of an exaggerated vesicular quality If the vesicular tension is less than normal, the breath sounds become purely vesicular, emphysematous If of bronchial origin, they are of a harsh piping quality, and are then termed *bronchial*. When the breath sounds are produced by a combination of bronchial and vesicular structures, they assume an intermediate quality. A cavity causes breath sounds of amphoric or of cavernous quality, depending upon the tension of its walls

Bronchial Breathing

This has already been described as high-pitched, harsh, tubular or piping in quality, and of great intensity The vesicular quality being entirely absent, expiration is as long as inspiration, and the intrarespiratory pause is lengthened: normally, this is heard over a large bronchus; *pathologically*, it occurs where the air vesicles have been put out of service and respiration in that part is being carried on only by the bronchi. This type of breathing is found in:

1. **Consolidation of the Lungs:** Whether the consolidation is caused by lobar pneumonia, pulmonary tuberculosis, hemorrhagic infarcts, new growths, pulmonary abscess or gangrene matters very little, so long as a sufficiently large portion of the lung is affected, thereby causing the respiratory air to travel in and out through these same tubes without being dispersed into the vesicular structures.

The intensity of this sound is enhanced because it is transmitted through consolidated air vesicles, and—since a solid substance transmits sounds more readily than does air—the bronchial breath sounds are thus better transmitted.

2 **Compression of the Lungs:** Portions of the lungs may be compressed to such an extent as to cause utter collapse of the air vesicles, thus leaving only the bronchi to carry on respiration. Compression of the lung may be caused by a large pleural effusion or by pneumothorax, a tumor, an enlarged heart, pericardial effusion or enlarged glands. The effusion occupying the lower portion of the chest must necessarily crowd the lung upward and toward the spine, thus causing bronchial breathing to be audible in that location, while no breath sounds can be heard over the effusion itself In pleural effusion or empyema following pneumonia the breath sounds often remain bronchial and frequently mask the presence of fluid.

3. **Bronchiectasis** may at times cause bronchial breathing

Bronchovesicular Breathing

This form of breathing occurs where there is blending of bronchial and vesicular structures Pathologically, it may be in evidence over:

1. **Small Consolidations** (found in pulmonary tuberculosis, bronchopneu-

monia and atypical pneumonia): The air vesicles immediately surrounding a consolidation become distended in order to compensate for the affected vesicles; when listening over a small consolidation, the blending of the sounds caused by both the consolidation and the distended vesicular structures are heard; hence a bronchovesicular quality.

2. **Deep-seated Consolidation:** Central pneumonia, where a portion of healthy lung overlaps the consolidation.

3. **Small areas of pulmonary atelectasis** due to any cause.

4. **First and third stages of lobar pneumonia,** before and after the occurrence of complete consolidation, or any other condition that causes partial infiltration of the air vesicles.

5. **Diffuse carcinomatosis,** enlarged bronchial glands and pneumonoconiosis.

Emphysematous Breathing (*Asthmatic Breathing*)

This form of breathing is always pathologic; it is never heard over a normal chest. It is of low pitch, wheezing in quality and low intensity. Expiration is a little longer than inspiration and the intrarespiratory pause is lengthened This form of respiration occurs in chronic emphysema and in asthma; it is heard over the entire chest on both sides, and, as a rule, is accompanied by numerous râles.

Emphysematous breathing is the result of chronic overdistention of the air vesicles, which causes them to lose their elasticity, and as a result, they are unable to collapse when necessary. The accompanying inflammation brings about an accumulation of small amounts of secretion in the vesicles and bronchioles The inspiratory air, being forced through the accumulated secretion and narrowed tubules, produces a wheezing sound and numerous râles. The expiratory sound is delayed and very much lengthened because the vesicles collapse slowly on account of their inelasticity, and also because of the plugging of the bronchioles, thus taking a longer time for the air to leave the lung structure. The inflamed and thickened bronchioles also act as good conductors of sound, thereby allowing expiration to be heard for a longer period than in the normal lung Emphysematous breathing resembles the sounds produced by the old-fashioned blacksmith's bellows.

Cavernous Breathing

Cavernous breathing is a low-pitched, hollow, distinctly "blowing" sound, resembling that which can be produced by blowing forcibly into the hollow of the cupped hands, the mouth being held wide open It is heard over a cavity with flaccid walls which communicates directly with a bronchus. Cavernous breathing may at times be audible over a consolidation overlying a very large bronchus or bronchiectasis.

Amphoric Breath Sounds

Amphoric breath sounds are harsh metallic blowing sounds, the pitch of which is much higher than in cavernous breathing A similar sound may be produced by blowing over the mouth of a china jar, or blowing forcibly into the hollow of the hand with the lips puckered as if to pronounce *oo.* Amphoric breathing can be heard over a very large, smooth, tense-walled cavity communicating with a large bronchus. It is also audible over a pneumothorax which communicates through a pleural fistula with a bronchus. The height of

the pitch depends upon the size of the resonating chamber.

Metamorphosed Breathing

This phenomenon was described by Seitz. It is a modified bronchial breathing; the first part of the inspiratory sound is harsh and bronchial, suddenly changing to a softened cavernous or amphoric sound and so remaining throughout expiration; it is due to the narrowing of a bronchus communicating with a cavity.

Vocal Resonance

Vocal resonance is the resounding tremor set up by the vibrations of the spoken voice as they are transmitted to the chest wall It is conducted to the listening ear as an indistinct rumble, the loudness of the rumble depending upon the intensity of the vocal resonance Vocal resonance is to auscultation what vocal fremitus is to palpation, both being produced by the same factor, namely, vibrations set up by the "spoken

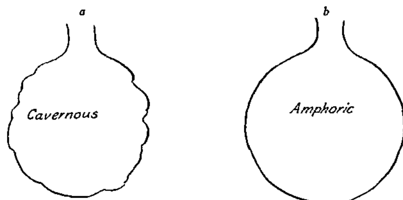


Fig. 11—*a*, Cavernous. *b*, Amphoric breathing.

Résumé·

Rhythm	Interrupted rhythm.	Slight deposits in the lungs
	Divided rhythm	Want of elasticity of lung
	Prolonged expiration	Want of elasticity in the lung, and consolidation
Quality:	Vesicular.	Uncomplicated lung
	Diminished.	Plastic exudations, want of elasticity.
	Absent breath sounds	Fluid or air in the pleura, large atelectatic area, massive pneumonia or plugging of the bronchi
	Emphysematous	Emphysema and asthma
	Exaggerated vesicular.	Vicarious respiration.
	Harsh	Moderate thickening of the bronchial mucous membrane
	Bronchovesicular.	Moderate consolidation or compression.
	Bronchial	Large consolidation, compressed lung and bronchiectasis.
	Cavernous	Cavity with flaccid walls.
	Amphoric.	Large cavity with tense walls.

voice." The same rules govern the transmission of sound in both instances.

Production of Vocal Resonance: The vocal cords are set into vibration by the spoken voice, which in turn sets into vibration the entire bronchial tree and the *entire bronchopulmonary column* of air. The vibrations are more perceptible in the large bronchi than in the smaller ones for three reasons: (*a*) Their cartilaginous structure; (*b*) their nearness to the chest wall, and (*c*) their caliber being large, they contain a greater amount of air.

Transmission of Vocal Resonance: Vocal resonance is, as an instance, unusually strong over the trachea because of its nearness to the larynx; its large caliber accommodates much air; its cartilaginous structure is a good vibrating medium and resonator; and the small *quantity of tissue covering the trachea* brings the vibrations closer to the ear

It is, therefore, evident that vocal resonance depends upon:

(*a*) *The amount of air in the part under examination*

(*b*) *The tension under which the vibrating air is held.*

(*c*) *The condition of the overlying structures through which the vibratory sound has to pass.*

(*d*) *Its distance from the larynx*

(*e*) *The condition of the vocal cords*

Vocal cords that do not vibrate will not produce vocal resonance.

Technic for Obtaining Vocal Resonance: Patient and physician place themselves in the proper auscultatory *position*. *The patient's chest is bared* and he is asked *to repeat* slowly in a deep loud voice, a consonating stock phrase, such as *one-one-one* or *ninety-nine, ninety-nine, ninety-nine,* while the examiner listens carefully, as he rapidly moves the stethoscope from one point to another, and compares corresponding points on both sides of the chest

Vocal resonance is heard with varying intensity over the different regions of the normal chest. It is more distinctly heard over the chests of persons having thin chest walls and deep, low-pitched voices. Vocal resonance is *generally louder in children than in* adults because of greater lung tension and a more resilient chest wall It is weakest in the aged because of the inelasticity of the lung and the nonresilience of the chest; louder in men than in women, and in the lean than in the fat.

It is more distinct over the right side than over the left, and anteriorly than it is posteriorly, excepting in the intrascapular region, where it is always very loud.

Regional Variations of Vocal Resonance[*]

Suprascapular Region

Right	Left
Generally weak near the outer half, somewhat more pronounced than on the left side Very loud near the sternal end, because of the presence of the trachea.	Weak to the left of the midclavicular line; louder as the sternal end is approached.

Suprasternal Notch

Very loud.

Infraclavicular Region

RIGHT	LEFT
Very loud, or increased vocal resonance because of the larger caliber of the right bronchus, more numerous bronchioles, and closer proximity of the trachea to the right lung.	Quite loud near the sternal end; of moderate intensity over the remaining region. The left bronchus is deep seated

Mammary Region

RIGHT	LEFT
Weak because of the pectoral muscles and mammary gland	Weak because of the pectoral muscles and mammary gland

Inframammary Region

RIGHT	LEFT
Absent except in its uppermost portion, or immediately above the liver.	Absent except in its upper portion above the stomach.

Suprasternal Region

Very distinct because of the underlying trachea, and the resilience of the sternum

Infrasternal Region

No resonance because of absence of lung tissue

Supraspinous Region

RIGHT	LEFT
Very loud	Not quite so loud as on the right side

Spinous Region

RIGHT	LEFT
Weak because of the scapula.	Weak because of the scapula

Interspinous Region

RIGHT	LEFT
Very distinct, particularly in the vicinity of the fifth dorsal spine	Very distinct, particularly between the fourth and sixth dorsal spines.

Infraspinous Region

RIGHT	LEFT
Weak	Weak

The supraäxillary regions of both sides present distinct vocal resonance

In the infraäxillary regions vocal resonance is weaker than in the upper regions.

Spine: Vocal resonance is very loud over the seventh cervical vertebra; the intensity of the resonance becomes weaker as the spine is descended; no resonance is perceived below the fifth dorsal spine, except in pathological conditions (SEE D'Espine's sign, p 335).

Pathologic Variations of Vocal Resonance

Because of certain pathological conditions the vocal resonance may become: (I) Increased; (II) diminished; (III) absent; (IV) altered

I. **Increased vocal resonance** may be due to:

(*a*) Any condition that will set more air in vibration.

(*b*) Any condition that will transmit the vibrating air with greater intensity.

(*c*) *A combination of* (*a*) and (*b*).

Increased vocal resonance is therefore found in:

1. Consolidation of the lung (the larger the consolidation, the more intense the resonance).
2. Infiltration of the lung.
3. A superficial lung cavity containing air and in direct communication with a bronchus.
4. Compensatory emphysema
5. Pleural adhesions.
6. A tumor or gland lying between a large bronchus and the chest wall.
7. Partially compressed lung.
8. Bronchiectasis.
9. Adhesive bands stretching from a bronchus to the chest wall, though the chest be *filled with an effusion* The adhesions act on the same principle as a telephone wire

II. **Diminished vocal resonance** may be caused by: (*a*) Conditions that fail to transmit the entire vibratory consonance; (*b*) conditions that fail to produce normal vibrations, and (*c*) a combination of (*a*) and (*b*).

Diminished vocal resonance is found in the following pathological conditions:

1. Thickened pleura, and thickened chest wall.
2. Small pleural effusions.
3. Chronic emphysema.
4. Laryngeal stenosis (partial).
5. *Edema of the glottis* (*partial*).
6. Tumor lying between the lung and the chest wall.
7. Edema of the lungs (moderate degree) and of the chest wall.

III. **Absence of vocal resonance** may be caused by conditions which fail entirely to transmit resonance, or which so compress the lung and bronchi as to hinder the production of resonance, and also in conditions where it is physically impossible to create resonance. *Absence* of vocal resonance is found in:

1. Large *pleural effusions* (*serum*, pus, blood or air)
2. Massive pneumonia.
3. Edema of the lungs
4. Deaf mutes.
5. Paralysis of the vocal cords.
6. Absence of lung structure (evisceration, diaphragmatic hernia, eventration).

IV. ***Altered vocal resonance*** is caused by pathological conditions in the lung which influence the vocal resonance as follows:

Bronchophony ("chest voice") This is the sound of the voice as heard by the listening ear when applied over a *normal bronchus during phonation. It* is a very loud indistinct humming sound, which seems to form under the examiner's ear, the intensity often being so great as to annoy the eardrum Bronchophony is normally heard over the trachea and *the large bronchi* during speech. *During an examination it may* be elicited by having the patient repeat *one-two-three, one-one-one, ninety-nine, ninety-nine, ninety-nine*, or any number of words, while the examiner listens with the stethoscope. To avoid error the patient should always turn his face away from *the ear of the examiner.*

Pathologically, bronchophony is found over:

1. Consolidation of the lungs (second stage of lobar pneumonia); large firm patches of bronchopneumonia; tubercular consolidations; retracted and com-

pressed lung above a pleural effusion; aneurysm or some other rapidly forming tumor which causes lung compression

2. A cavity adjacent to, or surrounded by, solid tissue or lung consolidation.



Fig. 12—Starting point for auscultating D'Espine's sign

3. Bronchiectasis (dilated bronchus), when superficially situated and empty.

4. Senile emphysema (rare).

Whispered Voice: Normally, *whispered voice* is transmitted only over the large bronchi, the trachea, over the spine of the seventh cervical vertebra, with lesser intensity over the second right interspace near the sternum, and in both interscapular regions opposite the spine of the scapula, the latter being points of vantage for reaching a bronchus The whispered voice is not transmitted over uncomplicated vesicular lung structure. Transmission of the whispered voice over vesicular structure indicates infiltration, partial consolidation or distention of the lung and is heard over small tuberculous or bronchopneumonic consolidations; it is also a sign of compensatory emphysema.

D'Espine's sign is the transmission of whispered voice over the spines of the spinal vertebrae. In the normal adult when auscultating over the spinous processes, it is found that the normal voice is not transmitted below the bifurcation of the trachea, fourth or fifth dorsal spines, and in young children below the seventh dorsal vertebra.

To elicit D'Espine's sign the patient is instructed to whisper *one-two-three* continuously, while the examiner ausculates over the spines of the vertebrae Auscultation is begun over the spine of the seventh cervical vertebra, and is continued downward over the spine of each succeeding dorsal vertebra until the whispered voice ceases to be audible

Pathologically the whispered voice may be heard as low as the seventh or eighth dorsal spines, and in rare cases, as low as the ninth dorsal spine. The presence of a positive D'Espine's sign is often an indication of peribronchial tuberculosis, thickening of the hili, central pneumonia, tumor or some other solid substance lying between a bronchus and the spinal column. In pulmonary tuberculosis the whispered voice is transmitted to a lower spinal level *than in health*

Pectoriloquy (chest speech): This is the transmission of articulate speech; it differs from bronchophony in that the latter signifies only exaggerated sound, while pectoriloquy stands for the transmission of words and syllables It often gives the listener the impression that the words are being whispered directly into his ear. Pectoriloquy may be ***spoken*** or ***whispered;*** whispered pectorilquy is of greater diagnostic value and more

readily distinguishable, for spoken pectoriloquy may often be confused with bronchophony.

Whispered pectoriloquy is brought out by having the patient whisper *one-two-three*, etc., at which time his mouth should be turned away from the examiner's ear. If a binaural stethoscope is used, care should be taken not to allow the rubber tubing to rest upon any portion of the patient's chest. The various parts under examination should be carefully compared.

Normally pectoriloquy is heard only over the trachea; pathologically it is heard over a superficial cavity communicating with a bronchus; less frequently, over dense consolidations surrounding a large bronchus and an open circumscribed pneumothorax freely communicating with a bronchus, and, at times also, over a compressed lung above a pleural effusion, or over the upper portion of a bronchus when the lower portion is compressed by a tumor

Egophony: This is a peculiar nasal sound, frequently compared to the bleating of a goat. It may be heard over consolidated or partially compressed lung when the subject speaks in a natural voice. This sign may be elicited over the upper layer of a pleuritic effusion, immediately below the line of percussion dullness, and over the fluid level of a cavity half filled with secretion; at times also, where a pleural effusion overlies a pulmonary consolidation. The absence of this sign does not exclude pleural effusion, nor does its presence necessarily indicate this condition.

Amphoric Voice Sound: This consists of a metallic, ringing, articulate voice sound, resembling the echo produced by speaking into a jar. It is heard over a large communicating cavity with tense walls; also over an open pneumothorax.

Baccelli's Sign (amphoric pectoriloquy): This sign is not trustworthy, and therefore, is of no especial value. Baccelli claims that the whispered voice cannot be transmitted through a purulent effusion, but that it may be heard over a serous effusion It is quite true that the whispered voice cannot be heard through a purulent effusion, but neither may it always be heard through an uncomplicated serous effusion

Résumé:

Normal vocal resonance.	heard	Over uncomplicated lung
Increased vocal resonance.	"	Over infiltration of the lungs, small consolidations, adhesive bands stretching from a bronchus to the chest wall.
Diminished vocal resonance	"	Over thickened pleura, small effusions, chronic emphysema
Absent vocal resonance.	"	Over pleural effusions, collapse of the lung, massive pneumonia
Bronchophony.	"	Over consolidation of the lung, bronchiectasis
Pectoriloquy (whispered)	"	Over a cavity; consolidation overlying a cavity of bronchus; bronchiectasis
Egophony.	"	Over compressed lung at upper level of pleural effusion, and above the fluid in a cavity.
Amphoric voice sound.	"	Over a cavity with tense walls

Phlegaphonia

Artificial vocal resonance is a procedure advocated by Scherwald, and is advantageously employed in dealing with deaf mutes, or with those who are suffering from aphonia. It will also prove useful for those who have just suffered a severe pulmonary hemorrhage or have vocal cord involvement, so that it is undesirable for them to speak; and in unconscious patients.

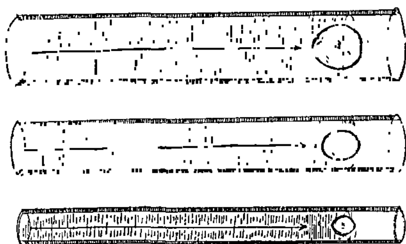


Fig. 13—Moist râles, large and small.

Technic: The patient keeps his mouth shut while an assistant gently taps upon the thyroid cartilage, the examiner meanwhile listening to the lungs. With sufficient practice this method will yield fairly accurate results, particularly in those deaf mutes in whom the thyroid cartilage can be repeatedly and forcibly percussed

Adventitious Sounds

These sounds should not be heard over the normal chest. The presence of any of these is an indication of some pathologic condition of the lungs, bronchi or pleurae. They include:

I. Râles or rhonchi.
II. Friction sounds.
III. Metallic tinkling or falling-drop sounds.
IV. Hippocratic succussion splash.
V. Water-whistle, or lung-fistula sound.
VI. Veiled puff.
VII. Posttussive suction.
VIII. Cough.
IX. Intermediate unclassified sounds

Before the character of an adventitious sound can be determined it is necessary to exclude those extraneous noises which may be produced upon the surface of the body by muscular contractions, involuntary twichings, hair crackling or bone crepitation

Four important points to be borne in mind by the examiner are:

1. To have the stethoscope properly adjusted so as to exclude external sounds.

2 Either to soften or to moisten the coarse hair upon the chest so as to prevent it from crackling.

3. To have the chest muscles thoroughly relaxed so as to prevent muscular sounds from being audible.

4. To instruct the patient to keep his shoulder joints immobile, so as to prevent bone crepitation.

I. Râles or Rhonchi

Râles are adventitious sounds heard during respiration; they are produced as the result of some morbid state of the respiratory apparatus; they may be numerous or scant; large and small; moist or dry; bubbling; crackling; whistling, or squeaking sounds and may be heard during inspiration and expiration.

Normally, in the respiratory system, *there is secreted just enough fluid of* a definite consistency to permit proper *lubrication. The various bronchi are of* a definite caliber, and the vesicular structures possess a definite elasticity; these conditions are responsible for the production of definite sounds during respiration, *i. e.,* the "normal respiratory murmur."

If, as a result of certain morbid conditions, too much secretion is thrown into the respiratory tract, it will hinder the free entrance and exit of air, so that the respiratory air is forced *through the* accumulated secretion, thus creating bubble; these bubbles are named *moist râles.*

Another condition may exist in which the bronchial mucous membrane becomes engorged; the caliber of the bronchi is reduced and becomes irregular, either because of the swelling of the mucous membrane or on account of adherent viscid secretion. The respiratory air, being forced through a narrowed or distorted vessel, produces abnormal whistling or grunting sounds, these sounds, because of their dry quality, are termed *dry râles.*

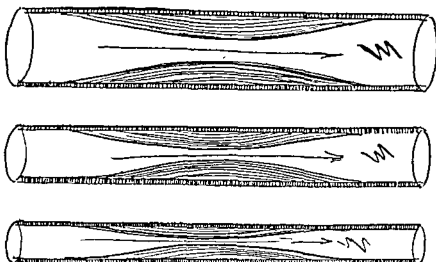


Fig. 14—Dry râles, large and small.

Râles are classified as *large and small* and *moist and dry* They may be inspiratory, expiratory or both. Their origin may be laryngeal, bronchial, vesicular or cavernous.

Large and Small Râles: A râle is spoken of as being large or small, depending upon the *caliber of the structure* from which it takes its origin. If it originates in the trachea, the larynx, a large bronchus or a cavity, it is a *large*

râle. If it originates in the small bronchi or the vesicular structures, it is a small râle. It is quite evident that large bubbles can be produced only in a large tube, while small bubbles occur in smaller tubes, therefore, the size of the râle depends upon the size of the tube.

Moist and Dry Râles: Râles are also classified as *moist* or *dry* according to the impression they convey to the ear

Moist râles usually resemble the sound produced by agitating soapsuds, or by Vichy water, or the bursting of bubbles which rise to the surface of water just beginning to boil Moist râles are spoken of as gurgling, bubbling (large or small), and subcrepitant; these are caused by a superabundance of secretion respectively in a communicating cavity, the bronchi, bronchioles and vesicles

1 ***Gurgling Râles, Gurgles or "Death Rattle":*** These are the largest and lowest pitched râles ever audible, and are often heard several yards away from the patient by the unaided, and even by the untrained ear. As the name indicates, they are large gurgles, caused by the accumulation of mucous secretion in the trachea The air, being forcibly driven through it both during inspiration and expiration, produces this succession of rattles. They usually occur in edema of the lungs and in terminal conditions

2 ***Cavernous and Amphoric Râles:*** These are gurgling sounds having a hollow metallic quality; they are heard over large pulmonary cavities communicating with a bronchus To produce these râles, the following conditions must be present: (*a*) The cavity must be large; (*b*) it must be about half filled with liquid secretion, the remaining part containing air; (*c*) the bronchus leading to the cavity must be unobstructed and reach below the level of the fluid.

These râles are heard both during inspiration and expiration, and are readily excited by coughing.

3. ***Bronchiectatic Râles:*** These râles closely resemble the cavernous variety, but somewhat lack their metal-

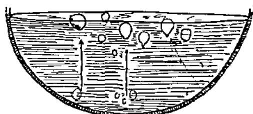


Fig. 15—Cavernous and amphoric râles

lic quality and also create an impression of distance. They disappear after a severe paroxysm of coughing if a large quantity of fluid is coincidentally expectorated. These râles are heard over bronchiectatic cavities containing a large amount of accumulated secretion.

4 ***Large Mucous Râles:*** These are loud, low-pitched, and of a bubbling character; they are heard over the course of large bronchi and indicate free fluid in these tubes, and are heard most frequently in chronic bronchitis

5. ***Medium-sized Bubbling or Submucous Râles:*** These râles are of a higher pitch and are more numerous than the large mucous râles; they are also heard over a large area, thus indicating involvement of a greater number of tubes of smaller caliber. These râles may be heard in the interscapular and supramammary regions, and may indicate the following conditions:

(*a*) A deep-seated bronchitis with mucoserous or purulent secretion.

(*b*) Pulmonary edema, the fluid having reached the level of the bronchi.

(*c*) *Pulmonary hemorrhage*, extending into the bronchi.

(*d*) *Inspiration of fluid into the lung* from immersion in water; during anesthesia in operations upon the throat; or other accidents; they are usually heard during inspiration.

6 **Subcrepitant or Fine Moist Râles:** These are the smallest of the

Râles

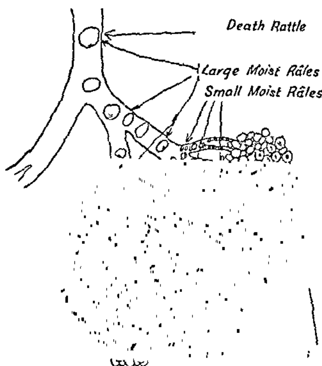


Fig. 16—Subcrepitant râles.

moist râles and are produced in the finest bronchioles and the vesicles; they have a peculiar quality resembling the bursting of tiny bubbles, or the sound produced by soapy water after agitation. These râles are usually heard over inflamed vesicular lung structure at the end of inspiration. *Mucous click* is a variety of subcrepitant râle; it occurs singly, resembling the sound produced by the separation of two fingers which have been previously glued together by a viscid substance. Mucous click is brought out more distinctly by coughing, and is frequently an early sign of incipient tuberculosis.

Subcrepitant râles are heard in: (*a*) Incipient pulmonary tuberculosis, apex. (*b*) Bronchopneumonia, found in many areas. (*c*) Lobar pneumonia, first and third stages, also adjacent to the consolidated area in the second stage (râle redux). (*d*) Pulmonary and hypostatic congestion in the interscapular region and at the base. (*e*) After hemorrhage at the seat of bleeding.

The *râle redux, or crepit redux*, of the older writers, is practically a subcrepitant râle. It is, as above indicated, found in the third stage of lobar pneumonia, and over healthy lung tissue bordering

the consolidated area during the second stage, and is probably caused by the overflow of fibrinous exudate into these portions

Dry râles occur as the result of contraction of the lumen of a bronchus, *which may be due to inflammatory thickening of its linings*, to adherent accumulated dried secretions or to partial compression of the bronchi from without by a tumor, adhesions, etc. In each instance, however, there is sufficient moisture to give the adventitious sound its inherent quality.

Some dry râles resemble a snoring sound, while others appear as a hissing or a whistling (sibilant) noise The dry râles originating in the large tubes are low pitched and snoring in character (*sonorous râles*) Those originating in the smaller bronchi are high pitched, hissing or whistling (*sibilant râles*). And those originating in the vesicles are the smallest dry râles (*crepitant*) and are caused by separation of the vesicles after having been glued together by a thin layer of viscid secretion. The presence of fine râles indicates acute inflammation

1. ***Sonorous Râles:*** These are large râles of a dry quality and low pitch, *i. e.*, the pitch is low in comparison to the smaller dry râle (sibilant), but is nevertheless much higher than that of any of the moist râles. The sonorous râle has a peculiar snoring or groaning quality. It is caused by conditions which produce inflammatory thickening of the mucous lining of a large bronchus, or a diminution of its caliber by constriction of the lumen from without, or by dry secretion adhering to its mucosa. Outside compression may be due to the pressure of a tumor, aneurysm, or an enlarged gland which encroaches upon a bronchus. These râles may be detected over the upper anterior portion of the chest and between

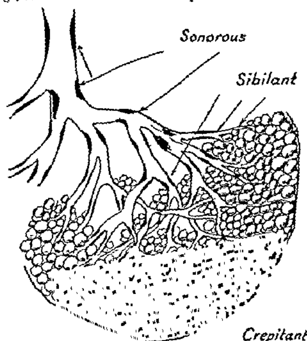


Fig 17—Sibilant râles.

the scapulae. As a rule, they are heard over a much larger area than their point of origin; at times they are loud enough to be heard at some distance away from the patient. When caused by external constriction, they are best heard immediately above and below the site of constriction.

2. ***Sibilant Râles:*** These are multiple, high-pitched, whistling, piping or squeaking sounds heard practically over the entire chest; they have a peculiar musical quality. The sibilant râles originate in the smaller bronchi and are caused either by partial obstruction of the lumen of these tubes by a viscid secretion, as occurs in bronchopneumonia, chronic bronchitis and emphysema; or by a spasmodic constriction of the lumen, as in asthma These râles may be heard both during inspiration and expiration.

3. ***Crepitant Râles:*** These are crackling sounds having a peculiar dry quality which may be simulated by rubbing a lock of hair between the fingers, or throwing salt upon a heated plate. Crepitant râles are the smallest râles usually encountered. As a rule, they originate within the air cells. Some clinicians believes them to be of extravesicular origin.

Intravesicular râles are caused by separation of the agglutinated vesicular walls. *Extravesicular râles* may result from the slow peeling off of the scantily fibrinated visceral pleura from each individual inflated vesicle. These râles are numerous at the end of inspiration, and are heard in pulmonary atelectasis, in incipient phthisis, in infarctions, and in edema of the lungs. They also accompany subcrepitant râles in pneumonia during the stage of resolution, and may be heard in bronchopneumonia Normally, a few crepitant râles may be heard either at the apices or bases of the lungs at the end of a full inspiration in individuals who are not in the habit of breathing deeply. After several deep breaths have been taken, however, these râles will cease to be audible It is often difficult to differentiate between crepitant and subcrepitant râles, and also between the subcrepitant and the smaller moist râles, for there is no fixed point where one may say that one variety stops and the other begins



Fig. 18—Crepitant râles.

The various fine râles may be schematically designated by the following signs:

. Crepitant x Subcrepitant
o Small, moist

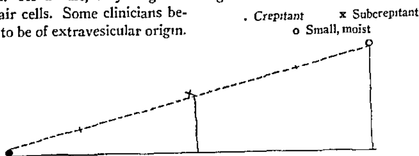


Fig. 19—Crepitant, subcrepitant, small moist chart.

Each subdivision is a little coarser than the one preceding, one variety gradually merging into the one which succeeds it, as illustrated in the diagram.

It may be the practice of one examiner to call all the râles from . to x *crepitant*, and all râles from x to o *subcrepitant*; while those beyond that point he may term *small moist râles* A second examiner may consider as *crepitant* only those râles which occur up to the first or second division in the first classification, while from that point to another point beyond the x he may term *subcrepitant*, etc.

It is obvious, therefore, that the point where one variety of small râle begins and the other ends, is both an arbitrary and a sliding one Usually each experienced clinician has in mind a definite point which serves him as a dividing line for the classification of small râles. In most instances, the recognition of small râles is sufficient for a diagnosis; only in special cases need they be definitely classified

Quality of Râles: Râles may be either *abundant* or *scanty*, their number depending upon:

(*a*) The quantity of fluid in the bronchi, air cells or cavity.

(*b*) The proximity of the affected part to the surface (facilitating transmission).

(*c*) The force of the respiratory current agitating the secretions

Numerous râles, therefore, indicate free communication between the diseased part of the lung or bronchi; if this be interrupted by the temporary impaction of mucus, the râles are either abolished or become very scanty, even though the parts be "loaded" with fluid secretion. Numerous and persistent large gurgling râles (bursting bubbles) are most frequently found in large pulmonary cavities containing much fluid; occasionally also, in smaller bronchi, when these are filled with secretion The less the amount of fluid in the respiratory tract, the scantier will the râles become, and the stronger will the inspiratory effort have to be in order to produce them.

Occasionally, in the presence of congestion, the secretion may be so scanty that only a few râles are heard at each inspiration; during several consecutive respirations none at all may be audible, their reappearance being facilitated only by coughing after expiration. At times also several inspirations may cause them to disappear completely. As before mentioned, in health a few scanty râles may be heard at the apices, the bases and the axilla; they are audible only during the first deep inspiration which causes separation of the alveoli and smallest size of bronchioles, and may disappear after the first distention The latter condition is usually found in subjects who are not in the habit of breathing deeply; also in those past middle life, in whom the edges of the lungs are somewhat atelectatic.

The *intensity* or *loudness* of a râle depends upon: (*a*) The abundance of the secretions; (*b*) the force of the respiratory act, (*c*) the size of the lumen of the bronchi containing the fluid, and (*d*) the nearness of the affected part to the chest wall.

It should be remembered, however, that when large râles are heard at a given spot on the surface of the chest they do not necessarily arise from the underlying lung. This is particularly true of the so-called *dry râles*. Therefore, when examining a chest, the area of greatest

intensity of a certain kind of râle should be noted. Because of the uncertainty of the origin of large râles, moist or dry, they are termed by some clinicians *indeterminate râles.* Small râles are not transmitted far beyond their point of origin.

Stage of Respiration in Which Râles Occur: Râles may be heard during inspiration alone, during expiration alone, during both inspiration and expiration, and during the respiratory pause.

If the râles originate in the smaller bronchi they are heard during the height of inspiration and at the beginning of expiration. But if there is sufficient secretion to clog not only the finer bronchi, but the larger air tubes as well, and respiration is carried on with sufficient force, the râles will be heard almost continuously during both inspiration and expiration, as in diffuse bronchitis Expectoration of the accumulated mucus after violent coughing causes a cessation of the continuous râles until the secretion reaccumulates.

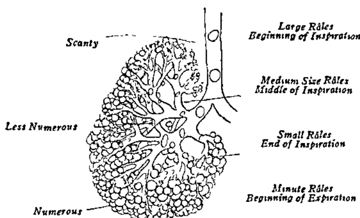


Fig. 20—Stage of respiration in which râles are heard.

Large râles occur at the beginning of inspiration and are few in number. The reason for this is obvious: The inspired air first passes through the large tubes which are fewer in number. The smaller râles are heard later in the inspiratory act, because the air reaches the smaller tubes later; they are more numerous because of the greater abundance of the smaller bronchi The smallest râles, crepitant and subcrepitant, have their origin in the alveoli, or the smallest of the bronchioles; therefore, their presence can be detected only at the very end of inspiration and the beginning of expiration.

Postexpiratory râles occur during the respiratory pause, and may be heard over large cavities and bronchiectases half filled with semimucoid secretion. The inspiratory and expiratory column of air agitates the fluid contained in the cavity to such an extent as to cause the bubbles to burst after the main column of air has left it. A like phenomenon may be seen at the ocean front, where the foam produced by the breakers continues to effervesce long after the wave has receded.

Râles are also classified according to their origin:

(*a*) Laryngeal	Moist	Gurgling.
(*b*) Tracheal	Moist Dry	Gurgling Sonorous
(*c*) Bronchial	Dry Moist	Sonorous Sibilant. Large, bubbling Small, bubbling
(*d*) Vesicular	Dry Moist	Crepitant. Subcrepitant. (fine soft crackle same as subcrepitant) Mucous click
(*e*) Cavernous	Moist	Gurgling, large, liquid.

Indeterminate Râles: Under this classification, the Army Medical School has included all large râles; that is, large and small moist râles, and sibilant and sonorous The teaching regarding the nomenclature and signs for râles at Fort Oglethorpe and the United States General Hospital No 16, at New Haven, Connecticut (the Army school for pulmonary tuberculosis during the first World War), was as follows:

. Crepitant râles, fine dry râles.
x Subcrepitant râles, finest of moist râles.
Indeterminate râles·
o Small mucous.
O Large mucous.
s Sibilant
S Sonorous.

The reason for classifying all the larger râles under the head of indeterminate is, as previously mentioned, because their point of origin is usually not accurately determined.

Significance of Râles: It is important to bear in mind that the existence of râles in the respiratory tract is indicative of an inflammatory process. Small râles, crepitant or subcrepitant, if persistent, are always an indication of *acute inflammation,* while large râles, moist or dry, are the result of *chronic inflammation.*

Differential Points Between Crepitant and Subcrepitant Râles

Crepitant Râles	*Subcrepitant Râles*
1 Dry crackling quality	1 Fine bubbling quality.
2 Numerous, an almost continuous crackling sound resembling the muffled explosion of a bunch of firecrackers, or the sound produced by treading on crisp snow	2 May occur singly or in smaller numbers, with sufficient pause between each râle to permit each one to be recognized as a distinct entity.
3 Uniform in size	3 Variable in size
4 Cough after expiration brings them out more plainly.	4 Brought out more plainly on coughing
5 Heard, as a rule, at the end of inspiration, during cough and at times during the beginning of expiration.	5 Occur toward the end of inspiration and at the beginning of expiration
6 Vesicular and extravesicular in origin.	6 Bronchiole and vesicular in origin

II. Friction Sounds

Normally, the pleurae are bathed by a serous fluid which acts as a lubricant, allowing free play between the visceral and the parietal surfaces. Certain inflammatory conditions may produce a deposit which causes the two surfaces to stick together lightly; therefore, when a "full breath" is taken, the pleurae are

intensity of a certain kind of râle should be noted. Because of the uncertainty of the origin of large râles, moist or dry, they are termed by some clinicians *indeterminate râles*. Small râles are not *transmitted far beyond their point of* origin.

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If the râles originate in the smaller bronchi they are heard during the height of inspiration and at the beginning of expiration. But if there is sufficient secretion to clog not only the finer bronchi, but the larger air tubes as well, and respiration is carried on with sufficient force, the râles will be heard almost continuously during both inspiration and expiration, as in diffuse bronchitis. Expectoration of the accumulated mucus after violent coughing causes a cessation of the continuous râles until the secretion reaccumulates.

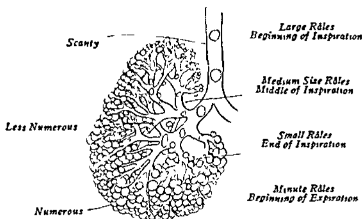


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Râles are also classified according to their origin:

thorax, and also over large cavities containing air and fluid. It may accompany the succussion splash, and can often be provoked by breathing, loud speaking, laughing, coughing, or by a change of position. The metallic tinkling may be due either to the dripping of fluid from the edge of the lung, or to the occasional bursting of a bubble upon the surface of the effusion. Both factors may be causative, because, in several instances, the differences in the qualities of the bursting bubbles and the falling drop have been detected in the same chest. This sound, also, may be heard over the normal stomach and bowel when inflated

IV. Hippocratic Succussion Splash

This is a splashing sound heard over the chest (either with the stethoscope upon the patient's chest or at some distance from the chest with the unaided ear) when the body of the patient is sharply shaken The condition can occur only when there is an accumulation of air and liquid in the pleura (hydropneumothorax and pyopneumothorax); it may also be heard over large cavities containing air and fluid Normally, a similar sound may be heard over the stomach and large bowel when these viscera contain a considerable amount of fluid and gas

V. Water Whistle Sound

This is described as a fine metallic bubbling or splashing sound heard when listening over a pulmonary fistula, such as that caused by puncturing a hydropneumothorax below the fluid level

VI. The Veiled Puff

This consists of a short hollow, whistling or puffing sound, heard at the end of inspiration, it is indicative of a sacculated bronchiectatic cavity

VII. Posttussive Succussion Splash

This is a "sucking-in," semisonorous sound, heard during inspiration after a paroxysm of violent coughing. It is often observable in cases of cavity with collapsible walls, communicating with a bronchus.

VIII. The Cough

(See: p. 95)

Much can be learned by auscultating the various regions of the chest while the patient coughs, because, by this procedure, the secretion in the air passages is more agitated than it is by respiration. In order to obtain the greatest amount of information through coughing, this act must be performed in a specified manner.

Technic: The patient is instructed to take a shallow inspiration followed by a deep expiration, at the end of which he is to give a short light cough *This cough should come from the diaphragm and not be a mere clearing of the throat.* When they first cough in this manner patients are often made dizzy, but frequent rest periods will obviate this unpleasantness, and in time they will carry on the "inspiration, expiration and cough" with ease for an extended period. In the presence of moisture in the lung vesicles, this form of coughing will bring out the crepitant and subcrepitant râles most prominently.

By auscultation of the cough, six points may be brought out which are valuable in diagnosis, and cannot be learned in any other way

1 After repeated coughing inspiration becomes deeper and the respiratory murmur louder.

2. Temporary obstruction of a bronchus or bronchi by numerous plugs is removed by coughing, particularly if the cough is followed by expectoration,

forcibly separated or torn apart; this is evidenced by a sharp pain, "stitch in the side," and on auscultation, a distinct *rubbing or grating sound may be heard over the area. Any condition causing* the surfaces of the pleurae to be roughened, so that one uneven surface glides over the other, will produce a peculiar rubbing or creaking sound.

Pleuritic Friction Sounds: These are rubbing, creaking, grating noises *heard during both inspiration and expiration* (loudest during the inspiratory act) over a limited area. The friction rub is best heard in dry pleurisy before the exudate is poured out; it disappears during the stage of exudation, and re-*appears toward the end of absorption. It is also heard in cases of pulmonary tu*berculosis, malignant disease, and syphilis affecting the pleurae, because of the production of uneven surfaces It is often quite difficult to differentiate between a friction rub and multiple râles; often the two phenomena may occur simul*taneously on the same side.*

Pleuropericardial Friction Sound: This is a typical friction sound differing only in time from the pleural friction sound. It is caused by contact of the roughened portions of the visceral pleura and pericardium as they lie in opposition to each other. This rub is heard during *inspiration because at that time the lung* border encroaches farthest upon the heart; it is also best heard during the cardiac systole because the heart is then moved upon the lung surface.

The systolic rub is constant and rhythmical, and cannot be influenced at will, *while the inspiratory rub may voluntarily* be made irregular by breathing faster or slower, or may cease entirely when the breath is held.

Differentiating Friction Rub from Râles

Pleural Friction Rub	*Râles*
1. Sounds very superficial to the ear.	1 Sounds more distant.
2. Strictly localized and cannot be heard two inches away from point of origin.	2 Not localized, but may be heard over a large area
3. Occurs only as a to-and-fro rubbing sound, depending upon the frequency of respiration	3 Sounds are multiplied, due to variety of râles.
4. The rub may disappear after numerous inspirations.	4 Not so affected by respiration.
5. Not influenced by coughing	5 May become either more numerous, or may cease after coughing.
6. Light pressure intensifies the sound; very hard pressure may stop it.	6 Not influenced by pressure.
7. Unilateral, accompanied by other signs of pleurisy.	7. Bilateral, associated with signs of bronchial affection
8. Often accompanied by a sharp pain and friction fremitus.	8 No sharp pain, no friction fremitus
9 Usually associated with distant breath sounds.	9 Usually associated with exaggerated breath sounds

III. Metallic Tinkling or "Falling Drop"

This is a fine resonant metallic tinkle, like the single stroke of a bell; it is of marked echoing quality, resembling the sound produced by the dropping of water into a partially filled cistern. This phenomenon is observed in hydropneumo-

General Résumé of Physical Examination of the Chest

Physical Condition	Inspection	Palpation	Percussion	Auscultation
Lung tissue normal or nearly so	Normal signs	Normal vocal fremitus	Clear note	Vesicular murmur or its modifications, normal vocal resonance
Lung tissue relaxed, loss of normal tension, moderate atelectasis, edema; deep congestion	Negative	Vocal fremitus increased	Vesiculotympanitic.	Bronchovesicular respiration; small mucous râles, vocal resonance increased
Consolidation of lung	Diminished respiratory expansion on affected side or locally.	Vocal fremitus increased.	Dull	Bronchial respiration, increased vocal resonance
Pleural effusion or tumor	Diminished movement on affected side.	Vocal fremitus diminished or absent	Flat	Absent respiration; sometimes distant bronchial breathing, absent voice; egophony rarely.
Increase of air in the vesicles, local or general emphysema, or cavities at particular points	Respiratory movement restricted generally or locally	Vocal fremitus diminished	Hyperresonance	Respiration feeble or cavernous; vocal resonance feeble or cavernous or exaggerated Mixed râles
Large cavity with elastic walls communicating with a bronchus	Diminished expansion over lesion.	Vocal fremitus diminished If air containing, vocal fremitus is increased	Amphoric, metallic, cracked-pot sound.	Respiration amphoric or metallic, cavernous, amphoric or metallic voice; whispering pectoriloquy
Air in pleural sac, open pneumothorax, closed pneumothorax Air under great tension	Greatly diminished movement	Absent vocal fremitus	Tympanitic metallic, amphoric coin test.	Absent breath sounds, absent vocal resonance

thus reëstablishing communication between the bronchi and the vesicular lung structure. The respiratory murmur, previously suppressed or indistinct, becomes clearer and its character is brought out more distinctly. Over consolidations and cavities, bronchial, bronchovesicular and cavernous breathing (depending upon the nature of the lesion) are often best heard after coughing.

3. Coughing frequently forces the secretion into the more confined spaces (the apices), thus increasing the number and intensity of the râles. Râles are heard with the greatest intensity during inspiration following the cough, occasionally also during the cough. Often, after coughing a number of times, the râles will become weaker, or disappear from one area to be heard in another. This is no doubt caused by the shifting of the secretion in the air passages, a phenomenon frequently encountered in diffuse bronchitis. Fine râles which are confined to one area, especially at an apex, and persist after coughing, are considered a pathognomonic sign of active pulmonary tuberculosis

4. Sibilant, sonorous and bubbling râles, in other words, râles of chronic inflammation, are brought out more clearly by *coughing after inspiration*, while crepitant and subcrepitant râles, the râles of acute inflammation, are best brought out by *coughing after expiration*. This latter method should be employed when examining for pulmonary tuberculosis and pneumonia

5. When auscultating over a consolidation the cough is exceedingly loud, almost ear-splitting in its intensity, while over a large superficial cavity it will have a metallic ring.

6. Cough when persistent, dry and not accompanied by râles may be due to reflex irritation from larynx or sinuses, or may be of nervous origin.

IX. Intermediate Unclassified Sounds

There is a variety of râle and other sounds which has thus far eluded classification, and these are therefore termed *intermediate râles*. They are crepitant, crackling, moist or dry sounds, which may be heard all over the chest during either part of the respiratory cycle, or throughout. They occur whenever there is moisture in the lungs, the bronchi and the pleurae. These sounds are not pathognomonic of any particular condition, though they are most often heard in bronchitis and asthma. Muscle sounds, bone crepitation, the "retrosternal crunching" described by Meyer Solis-Cohen, and other sounds that cannot be distinctly classified may be grouped under this heading

Muscle Sounds: Some individuals are able to contract their muscles so as to produce a succession of sounds not unlike small râles Often the fibrillary muscle twitching produced by coughing, or by a chill will serve to produce them. These sounds will cease as soon as the muscles are made to relax by a change of posture or by warmth. Muscle sounds heard at an apex are particularly confusing.

dition within the upper air passages, the trachea or the bronchi, and also by the presence of enlarged tonsils, sinus infections, focal infections, enlarged pendulous uvula, adenoids, congenital malformation of the trachea, or enlarged bronchial glands A foreign body in the bronchi or lungs may at times be the cause of chronic bronchitis Whooping cough, influenza and the exanthemata may leave their sequelae upon the respiratory organs so as to be the perpetuating cause of a chronic bronchitis *In the old* the continuous inhalation of irritating vapors, frequent exposure to wet and cold, and repeated attacks of acute bronchitis, pneumonia, cardiac decompensation, allergic conditions, focal infection and sinusitis may induce this chronic condition

Symptoms: These are cough which occurs in paroxysms, copious expectoration, absence of fever, and a history of long-standing cough.

Physical Signs: A person suffering from chronic bronchitis is usually emphysematous. *Inspection,* therefore, will reveal an emphysematous chest. *Palpation* will give evidence of diminished tactile fremitus throughout the chest. *Percussion* will elicit a hyperresonant note, except when associated congestion of the bases is present, in which case, impaired resonance or relative dullness is obtained over these areas On *auscultation* the examiner will hear low-pitched, prolonged inspiration, accompanied by low-pitched, prolonged wheezy expiration. The râles heard will be large and small, moist and dry. A profusion of all kinds of râles is usually audible in this class of cases, though the râles may disappear temporarily after the secretion has been coughed up

Fibrinous Bronchitis

Fibronous bronchitis (rare) is a chronic inflammatory condition of the bronchial tree, though at times it may be acute; it is characterized by the production of fibrinous casts of the bronchi.

Symptoms: These are similar to those of the ordinary form of bronchitis, except that the cough and dyspnea are exaggerated. Expectoration is scanty until the cast is brought up. The cough may occur in paroxysms, and is often accompanied by bloodstained expectoration.

Physical Signs: On *inspection* the patient appears to be very much distressed, and seems to have a mild degree of inspiratory dyspnea. Upon *palpation,* if the lumen of a bronchus supplying a large area of lung be plugged with fibrinous exudate, that area will be the seat of absence of tactile fremitus and diminished expansion. However, such an area is seldom large enough to give rise to these definite signs *Percussion* elicits nothing abnormal, unless a temporary atelectasis occurs, when impaired resonance will be elicited. *Auscultation* reveals a somewhat harsh inspiratory sound, with sibilant and sonorous râles

Foreign Bodies in the Bronchi

The presence of foreign bodies in the bronchi produces the signs and symptoms of chronic bronchitis. Inspiration of foreign bodies—especially by children —is not uncommon In the absence of a history, a positive diagnosis of this condition is possible only with the aid of the x-rays.

When the foreign body is actually in passage from the larynx downward to a point beyond the first bifurcation of the primary bronchi, the symptoms are those of strangulation, *i. e*, dyspnea, cyanosis,

CHAPTER XIV

Symptoms and Physical Signs of Diseases of the Respiratory System and Mammae

Diseases of the Bronchi

Acute Bronchitis

Acute bronchitis is an acute disease of the bronchi, characterized by a congestion of their mucous membrane, caused by the chemical and biological extension of irritation from the upper air passages, often following a rhinitis or a laryngotracheitis, inclement weather often predisposes to this affection. The larger bronchi are first affected. Affection of the smaller bronchi may be secondary to affection of the larger tubes. Further spread of the infection may cause bronchopneumonia. The condition is also found in association with influenza, measles, scarlet fever, and some of the other exanthemata and acute febrile diseases

Symptoms: These are retrosternal pain, hoarseness, cough, and often infracostal soreness; there may be a slight rise of temperature, though the temperature often remains normal

Physical Signs: *Inspection* of the chest is negative; the trachea and pharynx may be injected. Nothing abnormal is elicited by *palpation* and *percussion*, but on *auscultation* the respiratory murmur may be harsher than normal, and numerous large moist or dry râles are found along the large bronchi, which often disappear after cough and expectoration

Chronic Bronchitis

This is a chronic inflammatory condition of the medium sized and small bronchi, associated with destructive changes in their epithelial linings and sometimes with destruction of their mucous membrane. As a rule, it is a secondary disease. It is characterized by dyspnea, cough and various types of expectoration. Some patients cough through the entire year, others cough most during the change of seasons. Some cough during the night and others during exertion Acute exacerbation of a chronic bronchitis occurs frequently. Chronic bronchitis is often classified according to the type of expectoration:

1. A *superficial* type commonly seen in men past middle life who are of a gouty diathesis, or are suffering from general arteriosclerosis or renal disease, or have been emphysematous for an extended period. Cough is generally brought on by exertion. The expectoration may be thin or tenacious.

2. *Dry catarrh*, seen in elderly emphysematous individuals, the cough coming in paroxysms, with very tenacious and scanty expectoration.

3 *Chronic bronchitis* of young nervous individuals, more common in females, who have a chronic cough but do not present any other physical signs.

4. *Bronchorrhea* which, in addition to the leading symptoms of chronic bronchitis, presents a profuse, watery, and, at times, mucopurulent expectoration.

5. *Suppurative* or *fetid bronchitis*, in which the sputum is very fetid and resembles that obtained from bronchiectasis or gangrene of the lung.

Most cases of chronic bronchitis occur in those past middle life. *In the young* it may be caused by some irritating con-

increased tactile fremitus *Percussion* over the bronchiectasis when empty elicits a muffled tympanitic note The author's modified "coin percussion test" often gives positive results.

Coin Percussion Test: Technic for performing the modified coin percussion test: A coin placed over the suspected area and tapped upon with another coin, will elicit an increased metallic sound which can be heard by the examiner when listening at the open mouth of the patient; or, if the coin is placed upon the trachea and is percussed with another coin while the chest is being auscultated, the area where the coin sounds are most distinctly heard is the location of the bronchiectasis.

Other Signs: Small multiple bronchiectatic cavities will give rise to cracked-pot sound When the bronchiectatic cavity is empty, cavernous breathing, whispered pectoriloquy and bronchophony can be elicited by auscultation. When the bronchiectatic cavity is filled with secretion, absence of breath and voice sounds will be found When bronchitis is associated with bronchiectasis, the physical signs are those of the complicating bronchitis. An x-ray study may reveal the

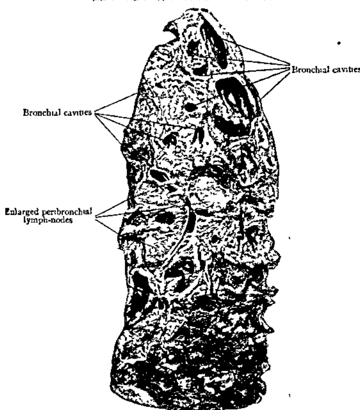


Fig 1—Bronchiectasis (*Da Costa*, W. B Saunders Co.)
(Jefferson Hospital Laboratories.)

protrusion of the eyeballs and retching. After the foreign body has found lodgment in one of the smaller bronchi, the symptoms and signs are those of acute or chronic bronchitis or they may simulate pneumonia.

Physical Signs: These depend on the location of the foreign body and the degree of obstruction it causes. Foreign bodies in the alveolar structures may cause no abnormal physical signs. Complete obstruction of a large bronchus results in atelectasis, and will cause the following signs on the affected side: Absence of expansion, lowering of the shoulder, flattening of the intercostal spaces, displacement of the heart towards that side, and dullness on percussion with absence of breath sounds. Partial obstruction or obstruction of a small bronchus may cause harshness of breath sounds, an expiratory wheeze and small bubbling râles over the affected portion of the lung.

A foreign body acting as a ball valve allowing the free entrance of air, but interfering with its exit, will produce signs of localized emphysema, *e g.*, localized increased expansion, hyper-resonance and exaggerated vesicular breathing, often associated with crepitant or sibilant râles. If pulmonary suppuration or an abscess has formed, the signs are those of suppuration, plus localized absence of fremitus and of breath sounds Foreign body in the trachea will be manifested by Jackson's three signs: (1) An "audible slap" as the foreign body is coughed up against the subglottic narrowing, (2) a "thud" palpable over the cricoid cartilage or trachea; and (3) an "asthmatoid wheeze" heard while listening at the patient's open mouth.

X-ray and fluoroscopic examination will readily detect an opaque body. The presence of a nonopaque body may be inferred from the usual signs of either partial or complete bronchial obstruction.

Bronchoscopic examination is often necessary for a definite diagnosis.

Bronchial Spirochetosis

This is a type of bronchitis caused by the spirocheta bronchialis. It may be acute or chronic. There is usually persistent cough with scanty, bloody and often fetid expectoration. The infection may spread to the lung causing gangrene or abscess.

Bronchiectasis

This is a saccular or cylindrical dilatation of the bronchi; it may be congenital or acquired, and occurs in one or both sides of the chest. Chronic bronchitis, tuberculosis, chronic sinusitis, whooping cough, and pulmonary infections are prominent etiological factors.

Symptoms: These are cough and expectoration, in severe cases, there may be dyspnea, general bronchitis and hemoptysis. The cough occurs in paroxysms and is often induced by change of position or by physical strain A sign frequently found in this condition is the expectoration of large quantities of foul-smelling secretion which takes place when the patient assumes a certain posture, or on arising in the morning The bronchiectatic cavity or cavities may thus be emptied several times a day, and in the intervals the patient will be fairly comfortable, and free from cough.

Physical Signs: *Inspection* usually reveals diminished general expansion, due to associated chronic bronchitis. *Palpation* shows that the tactile fremitus is increased when the bronchiectatic cavity is superficial and empty. The lung tissue immediately surrounding the enlarged bronchi may also impart a slightly

Physical examination elicits nothing characteristic besides the signs of acute bronchitis. The disease is characterized by its paroxysms of coughing, each paroxysm consisting of a number of short expiratory coughs followed by a long-drawn-in strangled "crowing" inspiration, the characteristic "whoop." During the paroxysm, the patient often becomes cyanosed. Severe and at times almost uncontrollable vomiting and hemorrhages may follow a violent paroxysm There is marked leukocytosis with a great increase of lymphocytes.



Fig 2—Pulmonary congestion (*Da Costa*, W. B Saunders Company) (Jefferson Hospital Laboratories)

Diseases of the Lungs

Pulmonary Congestion

This may be either active or passive. *Active congestion* may be due to some active inflammatory condition, infection, fever, or the inhalation of irritating substances, particularly irritant gases, such as phosgene, diphosgene, mustard or other gases, also to air of extreme temperatures. *Passive congestion* usually occurs as a result of some condition which interferes with the return circulation, dilatation of the right ventricle, mitral stenosis, or other conditions which cause heart failure. The principal seats of congestion are the bases or dependent parts of the lungs.

Symptoms: These are dyspnea, some cyanosis, irritating cough, and scanty, frothy expectoration.

Physical Signs: *Inspection* reveals moderate dyspnea with short, rapid, respiratory movements, cyanosis of the lips and finger tips, diminished expansion being observable throughout both lungs.

fibrosis and enlarged bronchi. These findings may be enhanced by lipiodol insufflation.

Bronchial Asthma

This is an acute paroxysmal dyspnea, generally expiratory in type which may occur at frequent intervals, and is often associated with chronic bronchitis and emphysema.

An asthmatic attack may be brought about by a variety of factors and may vary in different individuals Among such factors are the pollens from certain plants, house dust, certain proteins, disease of the Schneiderian membrane, nasal polyps, sinusitis, animal emanations, intestinal parasites, and other substances to which a particular individual may be allergic. Asthma also may be found in those suffering from pulmonary tuberculosis, heart disease, kidney and stomach disorders. Whatever the underlying factor may be, the condition is brought about by a spasmodic contraction of the bronchioles, which interferes with the exit and entrance of air to and from the lungs

Symptoms: During an attack the patient either sits erect or stands leaning against some object, grasping it firmly so as to bring into play the accessory muscles of respiration, and presents a characteristic appearance, *i. e*, the face is cyanosed, the eyes protrude, the superficial veins are prominent and perspiration is copious The respiratory movements are forced and of the up-and-down type; the patient has the general appearance of being strangled

Physical Signs: Between the attacks in early cases, there may be nothing definite; but after repeated attacks of asthma the patient may eventually develop chronic emphysema with its definite physical signs. For further details, see p. 924.

Hay Fever

This is a catarrhal condition of the upper air passages often extending throughout the entire bronchial tree, caused by some sensitizing substance, *i e.,* plant pollens. In many cases it is associated with asthma.

Physical Signs: These are like those of chronic bronchitis, superimposed by an acute coryza. The diagnosis is based upon the recurrence of the affection at a certain time of the year and its recurrence each year at precisely the same time. Skin sensitization tests will often reveal the specific cause

Our conceptions regarding the etiology and treatment of asthma, hay fever, the various allergic phenomena and certain skin manifestations—notably eczema and angioneurotic edema—have changed. The extensive investigations of the phenomena of anaphylaxis, allergy and protein-sensitization by such workers as Vaughan and Rosenow and the application of the findings of these investigators to the treatment of respiratory diseases by I. Chandler Walker and other clinicians, have wholly altered the general attitude of the medical profession, so that at present, asthma, hay fever, etc., are no longer classed as disease entities, but rather as symptoms of a constitutional affection

For further details, see p 925

Whooping Cough

This is an acute infectious catarrhal disease characterized by an inflammatory condition of the trachea and upper air passages. It is probably caused by the *Bacillus pertussis* of Bordet-Gengou. The disease occurs most frequently in young children.

expectoration, chills, fever and sweats The temperature is irregular, running from 99° to 103° and 104° F.

Physical Signs: These depend largely upon the size of the abscess and its location. If it is large, superficial and has persisted for any length of time, the following will be noted.

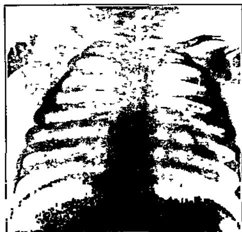


Fig 3—Lung abscess following pneumonia in a child of two years (Courtesy, Dr. Leon Solis-Cohen)

Inspection shows the patient to be emaciated and anemic; the lips and fingernails are cyanotic; respiration is rapid and expansion over the affected side is limited *Palpation* reveals absence of tactile fremitus over the affected part. If the abscess is in the lower part of the right lung, the "apex beat" will be pushed toward the left; if it be in the lower part of the left lung, the apex beat will be displaced to the right of the sternum. *Percussion* elicits dullness over the abscess, hyperresonance over the adjacent lung. On *auscultation*, before the abscess ruptures into a bronchus and empties itself, there is absence of breath sounds, or at best, very distant breath sounds over a limited area. After the abscess is evacuated, bronchial breathing can be heard over that area. An x-ray examination will usually confirm the tentative diagnosis of abscess, and in the case of a superficial abscess, the exploratory needle will make it positive. A bronchoscopic examination will at times help in the diagnosis of a pulmonary abscess when other methods fail When pus is expectorated it presents a characteristic fetid odor.

X-ray Findings: The lesions are usually single, but multiple abscesses may sometimes occur. The usual situation of the shadow is near one of the hili or toward the bases. They vary in size and though they may be well circumscribed, are usually irregular, the area of greatest density being in the center and fading out toward the periphery Cavitation generally occurs, and the cavity may contain air or fluid, or both The point of surgical attack is best obtained at the fluoroscope, by rotating the patient so as to determine the point of nearest approach to the lateral chest wall Simple multiple abscesses may be mistaken for metastatic malignancy and must be carefully differentiated

Pulmonary Gangrene

This is caused by decomposition of devitalized lung tissue as a result of bacterial putrefaction It may be localized or diffuse. It is caused by hemorrhagic infarcts; foreign bodies, tumors pressing upon the lung, pulmonary spirochetosis, focal infection or inflammatory processes of the lung, such as lobar and bronchopneumonia, or tuberculosis It may also occur as a complication in certain infectious diseases and in diabetes mellitus.

Symptoms: These depend largely upon the size of the area involved; if small, the most important symptoms are cough, fetid expectoration and extreme

On *palpation* over the congested areas, if the congestion is localized, slightly increased tactile fremitus may be elicited. Over the congested areas the *percussion* note is of higher pitch and resonance is impaired, while the areas adjacent to the hyperemic parts usually yield a hyperresonant note. *Auscultation* over the congested areas reveals bronchovesicular breath sounds. Vocal resonance is somewhat increased, numerous subcrepitant and other fine râles are heard, particularly after coughing. Over the areas adjacent to the congested parts, exaggerated breath sounds (puerile respiration) and a hyperresonant note are elicited.

Pulmonary Edema (Edema of the Lungs)

There are two varieties of pulmonary edema — *general* and *local* or *collateral.* Edema of the lungs usually follows congestion of the viscera, either active or passive. In congestion of the lungs there is an increased amount of blood in the vessels supplying and traversing the lungs. The increased pressure within these vessels produces congestion. When the congestion proceeds a step farther, serum transudes or exudes from these vessels and oozes into the interstitial structures and the alveoli of the lungs, causing edema. In *general edema,* both lungs are usually the seat of the affection The principal etiological factors are heart failure and general irritation caused by some mechanical, chemical or biological agent.

Local or collateral edema takes place adjacent to an inflammatory area or a new growth in the lung, such as the area adjacent to a pneumothorax, an abscess, a tuberculous or a syphilitic lesion, a malignant tumor or an aneurysm

Symptoms: Edema of the lungs may come on suddenly or gradually. The leading *symptoms* are dyspnea (each respiratory effort bringing up a quantity of frothy mucus), cyanosis, and often convulsions. If generalized edema is not rapidly relieved, death will soon result

Physical Signs: *Inspection* shows cyanosis of the lips and finger tips, and shallow respiratory movements, which are also feeble and rapid *Palpation* confirms inspection as to the respiratory excursions; tactile fremitus is usually diminished and the pulse is weak, feeble and thready. *Percussion* reveals impaired resonance. On *auscultation* the breath sounds are indistinct because of the presence of numerous large and small (moist) bubbling râles. The râles can be heard over the entire edematous area and often overshadow any other auscultatory findings which might otherwise be in evidence.

Pulmonary Abscess

This is an acute localized accumulation of pus within the lung substance, due to bacterial infection such as the streptococcus, diplococcus, pneumococcus, Bacillus of Friedlander, staphylococcus, spirocheta pallida, bacillus coli, and often also, to the amebae and certain other parasites An abscess of the lung may occur as the result of some localized inflammation arising from a penetrating wound of the lung, the aspiration of foreign bodies through the nose or mouth. and at times, after surgical operation in the buccal cavity, *i. e.,* tonsillectomy; or from an infection carried to the lung as a metastatic abscess. Pulmonary tuberculosis, unresolved pneumonia and foreign bodies frequently set up a localized abscess in the lung.

Symptoms: These are pain referable to the site of the lesion, cough, fetid

tactile fremitus; *percussion* yields hyperresonance; while *auscultation* reveals exaggerated vesicular breath sounds, both inspiratory and expiratory, which are a little harsher, but not quite so harsh as the bronchovesicular sounds. The ratio between inspiration and expiration is maintained as in normal breathing, though both are increased in length. Thus:

	Inspiration		Expiration
Normal respiratory ratio	3	.	1
Compensatory emphysema ratio	6	.	2

Differential Diagnosis

	Chronic Emphysema	Compensatory Emphysema
Inspection	Diminished expansion, barrel-shaped chest, weak apical impulse	Localized increased expansion
Palpation	Diminished tactile fremitus	Increased tactile fremitus
Percussion	Hyperresonance (low-pitched).	Hyperresonance (slightly higher pitched)
Auscultation	Emphysematous breathing, prolonged expiratory sound which is equal to the inspiratory sound (both being of a low-pitched and breezy quality); often numerous moist and dry râles Diminished vocal resonance.	Breath sounds resembling the puerile or exaggerated vesicular inspiration, and expiration twice as long as the normal, the ratio between inspiration and expiration being as six to two Increased vocal resonance.
Pathology	Overstretching, with loss of elasticity of the alveoli.	Stretching of the alveoli without any loss of elasticity; the elasticity of the alveoli is often much greater than the normal.

Pulmonary Infarction and Embolism

Pulmonary infarction causes collapse of a portion of lung which becomes blood-infiltrated It is due to occlusion of a pulmonary vessel by a thrombus or embolus It may be caused by subacute bacterial endocarditis, auricular fibrillation, phlebitis, and thrombosis of the femoral or pelvic veins following acute infections and surgical operation or trauma Large infarction may cause sudden death

Symptoms: If the infarct is large there will be pleural pain, cough, dyspnea, cyanosis, rapid heart action and fever.

Physical Signs: On *inspection* dyspnea, with limited expansion on the affected side, will be noted. *Palpation* will yield increased tactile fremitus over the infarcts; there will be dullness on *percussion,* and *auscultation* will reveal bronchial breathing, bronchophony and many moist râles. These signs will be demonstrable if the infarct is large, and is situated between a large bronchus and the surface of the lung. A small central infarct may be passed unnoticed during physical examination

A moderate-sized infarct away from a bronchus will present the following physical signs: *Inspection,* diminished expansion; *palpation,* decreased tactile fremitus; *percussion,* relative dullness and *auscultation,* distant breath sounds and moist râles over the infarct and exaggerated breath sounds over the healthy lung immediately adjacent to the infarct.

Pulmonary Arteriosclerosis

This condition is characterized by widespread sclerosis of the pulmonary artery or the smaller vessels. It may

fetor on the breath, very much resembling fetid bronchitis Extensive pulmonary gangrene will cause loss of weight, weakness and occasionally rise of temperature, often of a septic character. Small areas, particularly if centrally located, may escape detection by *physical signs,* the only clues being the cough and the extreme fetor of the expectoration and breath Large gangrenous areas will give signs similar to those of pulmonary abscess An x-ray examination may identify the lesion

Chronic Emphysema

There are three recognized varieties of emphysema: *Pseudohypertrophic, hypertrophic* and *interstitial* Emphysema (chronic) is due to atrophy of the alveolar walls with permanent distention of the air vesicles. An increase of intraalveolar air pressure, with possibly a congenitally defective development of the pulmonary elastic tissue, is necessary for the development of the pathological changes (Musser)

Pseudohypertrophic emphysema, called by Musser *acute vesicular emphysema,* is a rapidly developing condition of overdistention of the air vesicles, which sometimes takes place in asphyxia, asthma, whooping cough, or angina pectoris. *It is not a true emphysema,* as recovery or death ensues before atrophy of the elastic tissue can take place.

Hypertrophic emphysema is a condition where the retractility and elasticity of the lungs have diminished as the result of overdistention of the air cells, permanently enlarging the lungs The condition is commonly a secondary one and develops during the course of other lung diseases; it may then be due to the strain upon the alveolar walls imposed by constant coughing.

Interstitial emphysema is caused by wounds of the lungs, or rupture of the air vesicles by continued violent coughing, so that air is present in the interlobular and subpleural tissues It occurs most commonly in the upper lobes and anterior surface of the lungs.

Symptoms: The most prominent symptoms of emphysema are dyspnea (because of the inelasticity of the vesicular walls), cough and expectoration.

Physical Signs: *Inspection* will show a barrel-shaped chest, the anteroposterior diameter being greater than the transverse diameter; the shoulders are elevated, the neck is apparently short, the epigastric angle obtuse, and the scapulae lie flat upon the posterior aspect of the chest. Respiratory movements are limited on both sides, and the chest movements are of the up-and-down type. *Palpation:* The tactile fremitus is decreased, and the cardiac apical impulse is weak, at times wholly impalpable. *Percussion* yields hyperresonance throughout. *Auscultation* reveals emphysematous breathing (prolonged, low-pitched, wheezy inspiratory sound, the expiratory sound being as long, or longer, than the inspiratory). Vocal resonance is diminished, and the râles are large and small, moist and dry, and can usually be heard over the entire chest because of the associated chronic bronchitis

Compensatory Emphysema

This is an acute condition due to an overfilling of the air vesicles, causing the vesicular walls to distend, and thereby increasing their elasticity. *This condition arises when one part of the lung is obliged to compensate for another portion which is temporarily incapacitated*

Physical Signs: *Inspection* shows increased expansion; *palpation,* increased

areas of the lungs, more often at one apex or base than at the other. *Palpation* confirms the inspected sign of diminished expansion; tactile fremitus is, as a rule, diminished. However, increased tactile fremitus may occur when the deposits are close to a large bronchus. *Percussion* reveals relative dull-



Fig 5—Pneumonoconiosis.
(Courtesy, Dr. Leon Solis-Cohen.)

ness If the deposits are small, broncho-vesicular breathing will be heard on *auscultation*. If the deposits within the lung substance are large, and the bronchi are dilated, bronchial breathing will be audible, and if this condition is associated with bronchiectasis, cavernous breathing may be heard Râles, subcrepitant, small, bubbling and sibilant, often occur at the same site. Pneumonoconiosis, particularly if caused by dust particles, is often associated with emphysema. The physical signs are those of emphysema, plus small areas of relative dullness at the apices. For x-rays see Fig. 5 above.

Pulmonary Atelectasis (Massive Collapse)

Pulmonary atelectasis (pulmonary collapse) is due to an absence of air from the lung. An entire lung, or an entire lobe, or the greatest portion of a lobe may be involved; this condition may be caused by complete obstruction or compression of a bronchus, paralysis of a lateral half of the diaphragm, injury to the chest, foreign body in the chest and some unknown conditions. Massive collapse is at times seen after general, and rarely after spinal anesthesia, and occasionally in the newborn before respiration is thoroughly established

Physical Signs: The patient is usually dyspneic and cyanotic.

Inspection: The affected side is immobile; the intercostal spaces are narrowed and often retracted; the trachea and the heart are displaced to the affected side. The opposite side usually shows signs of compensatory emphysema.

Palpation: There is absent or diminished expansion; tactile fremitus is absent when the entire lung is collapsed, but may be increased when a collapsed portion of a lobe lies adjacent to a large bronchus.

Percussion: When total collapse is present a dull note is elicited, but when partial collapse is present or when associated with a partial pneumothorax then a tympanitic note is elicited. The diaphragmatic excursions are practically *nil*. The diaphragm on the affected side is drawn upwards.

Auscultation: When the atelectatic lung is in proximity to the mediastinum, bronchial breathing and increased vocal resonance are elicited; but when the col-

be primary in which the lesser circulation is affected, or secondary to syphilis, tuberculosis, bronchiectasis, and to prolonged hyperemia caused by pulmonary affections, cardiovascular disease, mitral stenosis and by marked chest deformities, *i. e.*, kyphoscoliosis.

Symptoms: These are cyanosis, dyspnea and orthopnea on slight exertion Cough may be either dry and hacking or be moist. The general symptomatology is that of chronic bronchitis.

Ayerza's Disease: This is a type of pulmonary arteriosclerosis with fibrosis. It is characterized by marked cyanosis, dyspnea, cough with expectoration, often hemoptysis, frequent headaches and chest pains. The blood count shows a definite polycythemia.

Asbestosis is caused by the inhalation of magnesium silicate.

Anthracosis is caused by a deposit of coal dust (coal miners' asthma).

Chalicosis and *silicosis* are due to the inhalation of particles of stone, and are usually found among potters, stonemasons, sand blasters, etc.

Siderosis is due to iron dust and is seen in steel grinders, mirror makers, goldbeaters, glass cutters, etc.

Organic dust causes a form of pneumonoconiosis found in grain handlers, threshers, backers, etc.

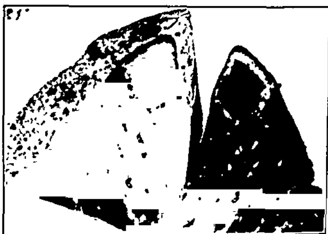


Fig. 4—Pulmonary infarct. (Jefferson Hospital Laboratories)

Pneumonoconiosis

This is a disease of the lung due to the inhalation and deposit of dust, mineral or vegetable, in the small bronchi, bronchioles and air vesicles. It is an occupational disease and is variously classified according to the kind of dust causing it.

Symptoms: The symptoms of all forms of pneumonoconiosis are similar to those of a foreign body encroaching upon the lungs.

Physical Signs: The physical signs depend upon the amount of dust deposited and also upon its distribution When a sufficient amount of dust finds its way into the lungs to produce symptoms which require medical attention, the physical signs are of a more or less distinct character. *Inspection* shows diminished expansion, particularly over the apices and often over other localized

the same as type V. The most prevalent types in adults are I, II, III, V, VII, and VIII. In children the more common types are XIV and VI, and in the aged, types III and VIII. The prevailing types of pneumococci often vary in different seasons and in different localities The symptoms and physical signs of most of the types are similar.



Fig 6—Lobar pneumonia; stage of gray hepatization (*Da Costa*, W B. Saunders Co) Jefferson Hospital Laboratories)

Symptoms: *The onset is sudden, often marked by a chill and pleuritic pain. The temperature rises rapidly, is of the continuous type and terminates by crisis or rapid lysis.* There is rapid breathing: the rate often depends upon the amount of lung involved, the severity of the toxemia and the amount of abdominal distention. The respiratory rate is high and out of proportion to the temperature and pulse rate. Cough may occur early but generally is a later manifestation of the disease The expectoration when present is tenacious, bloodstained or "rusty."

Physical Signs: *These depend largely upon the stage of the disease. Over the affected area the following will be found:*

lapsed lung is close to the chest wall and away from the mediastinum, then breath sounds are absent or distant and voice transmission is poor. Moist râles are heard over the atelectatic areas.

Small atelectatic areas may occur as the result of blockage of a small bronchus or bronchi. This may be found in bronchopneumonia, aspiration pneumonia, pulmonary tuberculosis, and other inflammatory conditions of the lungs and bronchi. Abnormal physical signs are often not demonstrable in this condition. Pulmonary atelectasis following anesthesia may cause physical signs resembling pneumonia. There is fever and many large and small moist râles.

The Pneumonias

The term pneumonia is generally understood to mean inflammation of the lung. In order to specify the type of inflammation certain adjectives are prefixed, such as bronchopneumonia, lobar pneumonia, interstitial pneumonia, etc. These terms denote in a general way the amount and kind of lung tissue involved by the inflammatory process; but in no way do these terms denote the etiologic factors responsible for the pneumonic processes. Physical signs may reveal the amount of consolidation present in the lungs, whether small or large, or single or multiple; the stage of consolidation, that is whether totally or partially consolidated and the presence or absence of accompanying involvement of the bronchi or pleurae. The data obtained by physical examination may indicate consolidation of the lung, but they are not sufficiently specific for the diagnosis of the type of pneumonia.

Clinically the pneumonias may be grouped in two general classes: (1) The pneumonias caused by the pneumococci, and (2) the pneumonias caused by other organisms and by physical agents. The clinical classifications of the pneumonias proposed by Rufus Cole and adopted by H. A. Reimann are the simplest and best suited for study.

(*a*) ***Clinical Lobar Pneumonia:*** The pneumonias caused by any one or more of the 60 or more types of pneumococci. This type usually involves the greater part of a lobe, an entire lobe or more than one lobe. Occasionally only a small portion of a lung may be the seat of consolidation.

(*b*) ***Clinical Atypical Pneumonia:*** The pneumonias caused by various cocci, bacilli (including Friedlander's bacilli), viruses, fungi, rickettsia, protozoa, metazoa, and those caused by physical agents such as the aspiration of foreign matter into the lungs. These types of pneumonia usually involve small portions of the lungs or several lobules. Occasionally a number of affected lobules may coalesce and involve the greater portion of a lobe or an entire lobe, or several large areas may show signs of total or partial consolidation.

Lobar Pneumonia

(Pneumococcic Pneumonia, Croupous Pneumonia, Pneumonitis, Lung Fever)

Lobar pneumonia is a primary disease of the lungs generally caused by the pneumococci Etiologically pneumococcic pneumonia may be classified into 60 different types, since there are 60 distinct strains of these microorganisms that may cause the disease. The number of strains is now generally given as 60 or more; type XXVI is probably the same as type VI, and type XXX is probably

atypical pneumonia. *Secondary atypical pneumonia* develops during the course of systemic infection in which the respiratory infection is incidental or a complicating feature. Atypical pneumonia is more frequently secondary than primary, and the lesions in the lungs are generally of lobular distribution; the chief physical signs are here described under the caption of bronchopneumonia.

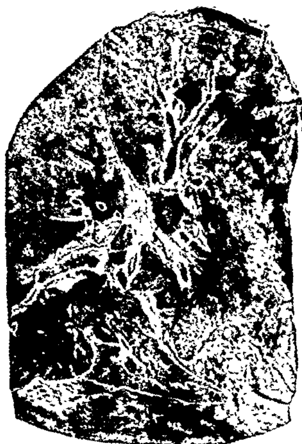


Fig 7—Lobar pneumonia; stage of red hepatization (*Da Costa*, W. B. Saunders Co)

Bronchopneumonia (Atypical Pneumonia)

Bronchopneumonia (lobular pneumonia, catarrhal pneumonia, capillary bronchitis) is an acute inflammatory condition of small portions or of several lobules of the lungs; the lesions may remain isolated or may become confluent

Etiology: It may be secondary to upper respiratory infection, or to measles, whooping cough, pyogenic infections and other systemic infections It may also result from the inspiration of foreign substances, from shock and occurs as a terminal manifestation in chronic diseases. As a *primary* infection it may be due to the invasion by the higher types of pneumonococci, streptococci, staphylococci, Friedländer's bacilli, influenza

	First Stage	Second Stage	Third Stage
Inspection:	Diminished expansion over the affected area.	Immobility of the affected part.	The immobile chest wall gradually assumes motion, as the disease is resolving
Palpation:	Increased tactile fremitus Occasionally tactile fremitus is absent	Greatly increased tactile fremitus.	Fremitus becomes less marked as the disease gradually resolves until the normal is reached
Percussion:	Tympany is often elicited during this stage because of the relaxation of the lung, as disease progresses relative dullness gradually merges into absolute dullness, displacing the tympanitic note	Dullness.	The dullness of the second stage gives way, first *to relative dullness, then* to impaired resonance, and finally to the normal note
Auscultation	Bronchovesicular breathing, increased *voice transmission*, crepitant and subcrepitant râles, particularly after cough. Occasionally there may be normal breath sounds or even an absence of breath sounds.	Bronchial breathing, bronchophony and whispered pectoriloquy; as a rule, no râles Occasionally there may be egophony When the lesion is centrally located signs of consolidation may be absent until late in the disease.	*Consolidation signs* gradually undergo modification, from bronchial breathing to bronchovesicular, then to normal breath sounds Voice sound changes from bronchophony to mere increased vocal resonance, and finally to the normal resonance. Subcrepitant and mucous râles

Adventitious Sounds Over the consolidated areas no râles are audible during the second stage of lobar pneumonia, but as resolution begins it is manifested by the return of subcrepitant râles, and *later by mucous râles; when resolution is complete, the râles disappear.*

Stages: The three stages of lobar pneumonia are often named in the order of their pathological sequence: (1) Stage of congestion; (2) stage of red hepatization, and (3) stage of gray *hepatization.*

X-ray Findings: Lobar consolidation casts a shadow of increased density, as a rule sharply defined, but gradually merging off into the noninvolved surrounding areas Small areas of involvement may occur in the base toward the pleura. There is no displacement of the heart. The process often spreads until the entire lobe is involved As resolution occurs the area of involvement breaks up into small areas of density with clearer radiability between. Often enlargement of the hilum shadow and parenchymal markings of the involved lung area persist for some time following the infection. Unresolved pneumonia casts a *similar shadow, and* a knowledge of the previous history is necessary to obviate confusion with intralobar empyema, particularly in a right sided lesion.

Atypical Pneumonia

Atypical pneumonia may be primary or secondary. *Primary atypical pneumonia* is described as a disease of the lungs in which the invading organism primarily *attacks the respiratory system* and the predominating signs are those of

the apex beat. Tactile fremitus may be slightly diminished, although often the fremitus is increased, because of adhesive bands stretching from a bronchus to the pleura *Percussion* usually elicits dullness or relative dullness over the affected portions, depending upon the size of consolidation; hyperresonance or tympany may be elicited near the angle of the scapulae, and close to Louis' angle. On

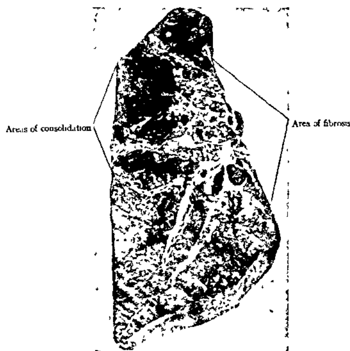


Fig 9—Chronic interstitial pneumonia (*Da Costa*, W. B. Saunders Company)

auscultation, the breath sounds are usually diminished in volume and are distant. When the bronchus supplying a portion of lung is clogged, no breath sounds are audible over that part However, if an associated bronchiectasis exists, then bronchial breathing is heard over that area. Vocal resonance may be diminished or increased, depending upon the amount of adhesions, the thickness of the pleura, and the condition of the bronchi.

Friedländer's Bacillus (Bacillus Mucosis Capsulatus) Pneumonia

This may be of lobar or lobular type. It is a comparatively rare but severe type of pneumonia affecting chiefly elderly persons

Symptoms: These are similar to the severe types of pneumococcic pneumonia Prostration comes on early. The sputum is thick and stringy containing blood, mucus and pus.

The physical signs are variable depending upon the amount of lung tissue involved. Often there may be signs of consolidation in one part of the lung and signs of congestion or of suppuration in

viruses, and by a host of other microorganisms of a single strain or of a combination of various organisms and strains

Symptoms: These are increased temperature, rapid breathing, cough and expectoration

Physical Signs: On *inspection* the respirations will be observed to be *rapid*

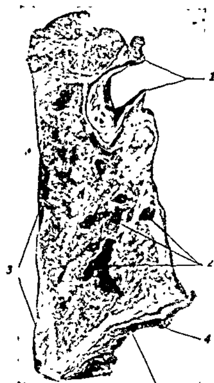


Fig. 8—Bronchopneumonia 1, Site of aorta; 2, exudate in bronchi; 3, pneumonic infiltration; 4, thickened pleura. (*Da Costa*, W. B Saunders Co.)

and shallow and the chest expansion to be diminished. *Palpation* will elicit increased tactile fremitus, both over the consolidation and the portion of lung immediately adjacent to it, and *percussion* will yield impaired resonance when the areas of consolidation are small, relative dullness over large consolidations and dullness when a number of consolidated areas have become confluent and occupy the greater portion of a lobe.

Auscultation will reveal bronchovesicular breathing and increased vocal resonance over small or moderate sized *consolidations*. Over large areas of consolidation, bronchophony and bronchial breathing will be heard. The pathognomonic signs of bronchopneumonia are *subcrepitant, crepitant and often other* types of moist râles over several areas, usually at the bases of the lungs, though any other portion of the lung may be affected by the disease. The râles occur early in the disease and remain throughout its course, until the lung has resumed its normal function. In the early stages, before any other signs are manifested, crepitant and subcrepitant râles can be brought out by cough, particularly when the cough follows expiration.

X-ray Findings: In bronchopneumonia there are multiple areas of shadows of uniform density scattered throughout the lobes involved. They are generally situated near the course of the larger bronchi and are rather hazy in outline, often giving the appearance of a powder puff. They must be differentiated from multiple abscesses.

Chronic Interstitial Pneumonia (Cirrhosis of the Lungs, Fibroid Induration of the Lung)

Fibroid induration of the lung is a primary or secondary chronic disease of the lung characterized by a deposit of fibrous tissue in the lung substance, and may be associated with chronic pleurisy.

Physical Signs: *Inspection* over the affected part shows the chest wall to be retracted, the interspaces are sunken, the shoulder droops and the spine is curved with its convexity toward the healthy side. The heart is drawn toward the diseased side. *Palpation* confirms inspection as to limited motion and the position of

(*c*) Intraluminal changes[1] These consist of a hemorrhagic, and at times an edematous, fibrinous exudate which later undergoes organization, with the formation of casts of loose fibrous tissue within the bronchioles. (*d*) The presence of a hyaline pseudomembrane.[2] The presence of a pseudomembrane was found covering the respiratory bronchioles, obstructing the mouths of, but not actually filling, the alveolar structures. However, such a membrane is also found in other types of pneumonitis. (*e*) The presence of Aschoff bodies. Only two investigators,[3, 4] reported their findings of these bodies, all others failed to discover them in the alveoli or other structures of the lungs

Diagnosis: The diagnosis of rheumatic pneumonia is based upon the abrupt onset with extreme respiratory difficulty, the dry hacking cough, the bloody or bloodstained sputum free from pathogenic organisms, the scarcity of abnormal physical signs in the lungs, and the x-ray findings of a diffuse, bilateral, nonsegmental infiltration of the lungs These findings in a person suffering from rheumatic carditis suggest the so-called "rheumatic pneumonia."

Prognosis: The prognosis of rheumatic pneumonia is extremely grave All the reported cases of this disease terminated fatally.

Lipoid-Cell Pneumonia[5] *(Lipoid Pneumonia)*

Lipoid-cell pneumonia is an aspiration pneumonia, occurring chiefly in debilitated children It is caused by the prolonged use of oily nose drops which find their way into the bronchi and the alveoli of the lungs, or by the inhalation, during feeding, of oily substances, such as cream cod-liver oil, mineral oil, or other fatty substances.

Clinical Course: The onset may be sudden or gradual with cough, rapid respiration, and a moderate rise in temperature. The mother or attendant may recall an incident where the baby strangled during the administration of cod-liver oil or other fatty substance, or there may be a history of the use of oily nose drops over a prolonged period. The general course may be acute or chronic.

Physical Signs: The physical signs and the roentgenographic findings are those of diffuse bronchitis or bronchopneumonia The infant gradually loses weight and is restless

Pathology: The alveoli and the alveolar cells contain fat globules (foam cells), and there is polymorphonuclear infiltration and inflammatory changes in the bronchial mucosa.

Diagnosis: This depends upon the history of having used oily nose drops or of having inhaled fatty substances, cough, rapid respiration, failure to gain weight, and signs of bronchopneumonia. The bronchial secretion or stomach washings may contain "foam cells" This condition is seldom diagnosed ante mortem It should be differentiated from tuberculosis by the tuberculin test, and the examination of the bronchial secretions

Prognosis: The prognosis in lipoid-cell pneumonia is variable. Robust children are more likely to survive than the asthenic ones Those who become sick because of the use of vegetable oils are more likely to survive than those who had used animal fats. Secondary infection also plays a part in the prognosis.

[1] Paul, J R Medicine, 7 383, 1928
[2] Masson, P, Ropelle J L. and Martin P Ann d'anat path, 14 359, 1937
[3] Frazer, A D Lancet, 1 70, 1930
[4] Gouley, B A, and Eiman, J · Am J M Sc, 188 359, 1932
[5] Laughlen, G F Am J Path, 1·407, 1925

another part. Pulmonary abscess is a common sequel.

*Rheumatic Pneumonia**

This is a term applied to certain acute changes occurring in the lungs of patients suffering from rheumatic heart disease. *Its occurrence* as a specific entity is a controversial subject. During the past two decades, a number of competent clinicians have presented case reports under the term "rheumatic pneumonia," describing the clinical course, the roentgenographic picture, and the autopsy findings Other investigators[2] deny the specificity of these findings and are opposed to the term "rheumatic pneumonia" as a distinct entity.

Mode of Onset: In the so-called "rheumatic pneumonia," the onset is abrupt with profound respiratory distress Dyspnea, often orthopnea, and cyanosis develop rapidly. There is a dry hacking cough which later becomes productive, and may be either blood-streaked or grossly bloody. Pleuritic pains may develop in any part of the chest; these are inconstant and fleeting. The temperature becomes elevated and is irregular, ranging from 100° to 103° F. or higher

Physical Signs: The physical signs, aside from the rapid and labored breathing, do not reveal any definite lung lesions. The heart shows evidence of cardiac failure which does not respond to digitalis, to the mercurial diuretics, or to bleeding

Laboratory Findings: *The leukocytes* may range from 14,000 to 26,000, and the polymorphonuclears from 75 to 93 per cent. Sputum *examination, by* smear and culture, fails to show any specific organisms.

X-ray Findings: The roentgenographic findings of the lungs resemble those occurring in cardiac decompensation. Seldin, *et al.*,[1] describes three types of roentgenologic findings. (*a*) vascular engorgement, that is undue prominence of arterial shadows throughout both lungs, (*b*) *pulmonary congestion as evidenced* by the appearance of coarse, fuzzy, arborizing, linear shadows radiating from the hilar regions; (*c*) pulmonary edema which is characterized by "a diffuse, moderately dense, fluffy or hazy parenchymal infiltration which is usually bilateral, multilobar, and often assumes *a 'butterfly' distribution with a peripheral* clear zone of emphysema and with varying degrees of obliteration of individual vascular shadows" This type of picture is usually obtained when cardiac failure *is associated with rheumatic pneumonitis*

Pathology: On gross examination, the lung has an "indiarubber-like" resistance, and its surface is mottled with focal hemorrhages. Microscopically the changes *within the lungs are of various* types as described by several authors: (*a*) Vascular changes.[3] These are deposits of fibrin within and about the systemic and pulmonary arterioles, with destructive changes and cellular *reaction* in the vessel wall and perivascular infiltration by inflammatory cells which later undergo organization (*b*) Alveolar wall changes.[4] There is thickening of the alveolar walls *by the proliferation* of endothelial cells, some fibroblasts, and occasional polymorphonuclear leukocytes

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* [illegible] of this subject, [illegible] 5, and [illegible] 947.

[2] [illegible] Arch Int Med, *68:1074, 1941*

[3] Von Glahn, W. C, and Pappenheimer, A. M : Am J Path, 2:235, 1926

[4] Naish, A. E. Lancet, 2:10, 1928

virus or by influenza B virus. There may be other types of influenza viruses not as yet identified As a rule influenzal pneumonia is caused by secondary invaders that grow in symbiosis with the influenzal virus The most frequent invaders are the *Hemophilus influenzae*, the pneumococcus, the Streptococcus hemolyticus, and the Staphylococcus aureus. The less frequent invaders are Streptococcus viridans, Staphylococcus albus, Micrococcus catarrhalis, Friedländer's bacillus, and meningococcus. However, the influenza virus alone is also capable of invading the lung and causing considerable bronchopulmonary reaction.

Clinical Course: Three types of influenzal pneumonia are described·[1] (1) A fulminating, rapidly fatal form in which pneumonia is present at the onset This form is comparatively rare; it is generally found in severe pandemics The onset is abrupt with *cyanosis and dyspnea*, hemorrhagic edema of the lungs appears rapidly, and the patient is in extreme distress Death may occur within twenty-four to forty-eight hours. (2) A progressive form in which signs of pneumonia begin to develop on the second to the fourth day of the onset of influenza and before the temperature has begun to decline (3) A late form in which pneumonia suddenly develops after the apparent recovery from the influenzal attack on the fourth to the tenth day after its onset With the exception of the onset, the clinical course of forms 2 and 3 are similar

Symptomatology: The temperature rises to higher level with *the onset* of pneumonia; it may range between 102° and 105° F. and is irregular Various aches and pains continue, cyanosis becomes more marked, and the respiratory rate increases. The cough becomes aggravated, and the expectoration may be scanty or profuse, mucoid or purulent, or bloody, and may be of pinkish or yellowish *green color. Pleuritic pain or a sense* of sternal oppression may occur during the early stages of the disease.

Physical Signs: Cyanosis and dyspnea continue until the crisis or lysis. The pulse is slow and out of proportion to the height of the temperature In fatal cases the pulse rate becomes rapid during the last thirty-six hours. The respiratory rate varies with the severity of the disease When the lesions are centrally located, examination of the lungs may reveal but few signs of the pneumonic process In the presence of superficial lesions, there may be impaired resonance and bronchovesicular, and at times bronchial, breathing Numerous moist râles over *the affected areas* are heard throughout the disease. The lesions are usually patchy and are often migratory; they are bilateral and occur most frequently at the bases

Laboratory Findings: In the blood count, the leukopenia of the primary disease often continues throughout the pneumonic process, polycythemia may develop in severe cases with marked cyanosis The blood culture is usually sterile The sputum may contain the secondary invaders The influenzal virus may be isolated from the sputum by an elaborate technic

Complications: These depend upon the type of the invading organism. Among the *complications occasionally encountered* are acute sinusitis, otitis media, purulent bronchitis, bronchiectasis, pulmonary abscess, empyema, interstitial emphysema, mediastinitis, and pneumo-

[1] Sydenstricker, U P.· In Cecil's Textbook of Medicine, W. B Saunders Co, p 10, 1945

The prognosis is less favorable when the secondary invaders are of a virulent strain of pathogenic micro-organism Many of the affected children die; those that recover do so by slow stages

Viral Pneumonia (Virus Pneumonia)

Virus or viral pneumonia may be divided into two categories: (1) primary atypical (virus) pneumonia of unknown etiology; (2) pneumonias associated with specific viruses.

PRIMARY ATYPICAL (VIRUS) PNEUMONIA OF UNKNOWN ETIOLOGY

During the past decade, a peculiar type of bronchopneumonia has become prevalent both in the United States and in England Dr. Hobart A Reimann,[1, 2] after an exhaustive study of this disease, named it "viral" or "virus pneumonia"

Symptomatology: The incubation period appears to be from one to twenty-one days. The onset of the disease is acute with temperature ranging from 99° to 105° F, and there is malaise, chilliness, sweats, and headache A distressing dry, nonproductive, uncontrollable, paroxysmal cough, which is more severe during the night, develops early in the disease. Substernal soreness and pain of the abdominal muscles result from the incessant coughing. The dry hacking cough may persist during the entire course of the disease, or after several days it may become productive and less painful

Physical Signs: On physical examination of the chest, there is a paucity of signs, the respiratory rate may be normal or only moderately increased. On percussion small areas of dullness may occasionally be demonstrable. Auscultation usually reveals, over various areas in the chest, small moist râles at the end of inspiration The pulse rate is seldom affected unless the temperature is high, or the patient has become exhausted from coughing.

Laboratory Findings: The sputum is free from pathogenic organisms. The blood count shows a normal red and white cell count. Occasionally there may be a slight increase or a decrease in the number of leukocytes. The sedimentation rate may be somewhat elevated The urine is normal

Roentgenologic Examination: The x-ray findings are out of proportion to the scarcity of physical findings. The x-ray plate will show soft hilar shadows extending toward the periphery of the lungs In the majority of patients, the infiltration occurs in the lower lobes

Course: The course of the disease is variable It may run from a few days to five or six weeks or even longer. I found that the patients who have a subfebrile temperature (99° to 100° F) often run a protracted course of from five to ten weeks, while those having a high temperature (103° to 105° F.) may recover in from three to six days. In many instances the temperature suddenly remits after the first twenty-four to thirty-six hours and is followed by a secondary rise which continues until the crisis is reached.

Prognosis: The prognosis of primary atypical viral pneumonia, barring complications, is usually good. The percentage of fatalities is low.

PNEUMONIAS ASSOCIATED WITH SPECIFIC VIRUSES

Influenzal Pneumonia: Influenzal pneumonia may be caused by influenza A

[1] Reimann, H A The Pneumonias, W B Saunders Co, p 253, 1938

[2] Reimann, H. A.: J. A M A, 111 2377, 1938

emphysema, asthma and congestive heart disease; (*c*) massive atelectasis and bronchial occlusions, and (*d*) inhalations of irritating dusts, vapors, gases or other substances that may cause repeated respiratory infections Pulmonary fibrosis may affect the entire respiratory system or it may be confined to one lung

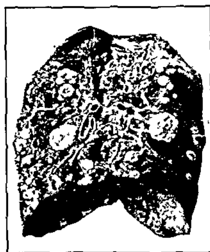


Fig 10—Multiple carcinomata of the lung (Philadelphia General Hospital)

or to one portion of a lobe, depending upon its etiologic factor The symptoms in massive fibrosis are dyspnea, cough, expectoration, a tendency to cyanosis and to cardiac weakness. The physical signs depend upon the degree and extent of involvement.

Inspection will reveal some dyspnea with limited chest expansion and often retraction of the chest wall *Palpation* may elicit diminished fremitus when associated with thickened pleura or with emphysema, and increased fremitus when the lung is solidified *Percussion* will yield various degrees of dullness, and on *auscultation* there is usually found various types of râles and often bronchovesicular breathing Clubbing of the fingers and polycythemia are frequently found in chronic massive pulmonary fibrosis.

Neoplasms of the Lungs

Tumors of the lung may be malignant or benign, single or multiple. Carcinoma and sarcoma are the malignant neoplasms most often encountered. The tumors may be primary or metastatic. They may originate in a bronchus, the lung, the mediastinum, or in some distant part of the body.

Symptoms: The symptoms are pain, dyspnea, cough and bloody expectoration, pleural effusion which is often bloody, and associated pressure signs.

Physical Signs: *Inspection* and *palpation* show limited expansion over the affected part, and there will be diminution or absence of tactile fremitus, if the tumor lies in close contact with a bronchus. Cyanosis and distended superficial veins are often noted upon the upper part of the chest wall *Percussion* elicits dullness, not movable, unless associated with effusion. *Auscultation* will reveal the absence of breath sounds if the tumor lies between the lung and pleura, because of the slight compression. If the tumor is adjacent to a bronchus and [illegible]

X-ray Findings: *Primary Malignancy* Primary malignancy of the lung is unilateral, carcinomatous in nature, and of the nodular or infiltrating type The situation of the former is usually at the hilum and consists of small sharply-defined nodules of medium density, possibly with striæ radiating into the adjacent parenchyma In the infiltrating type, the tumor may arise from the bronchus infiltrating along its branches

thorax. A postinfluenzal bradycardia may persist for a long period after recovery.

Prognosis: This is variable, depending upon the severity of the infection. Persistent cyanosis and tachycardia are bad omens

Psittacosis Virus Pneumonia: The psittacosis virus is transmitted to man by members of the parrot family, canaries, finches, pigeons, and their nests, and occasionally by chickens who were in contact with infected birds. The incubation period is from eight to fourteen days.

Clinical Course: The onset of the disease is abrupt, with malaise, anorexia, chills, backache, and headache During the height of the infection there develop restlessness, insomnia, delirium, and often the typhoid state Nosebleed is fairly common. The temperature rises rapidly and is more or less of the continuous type In favorable cases it may terminate by lysis during the third week

Physical Signs: The pulse is relatively slow, the respiratory rate is moderately increased. A decided increase in the pulse and respiratory rates is a bad prognosis In some cases rose-colored spots develop upon the abdomen. The examination of the lungs may reveal signs of lobular consolidation which starts at the hilum and spreads towards the periphery. The distribution of the lesions resembles influenzal pneumonia.

Laboratory Findings: The blood count at the onset shows a milk leukocytosis, later in the disease there is a moderate leukopenia. The sputum is tenacious but not rusty. Injection of some of the sputum into a mouse will usually reveal the psittacosis virus. The complement fixation reaction is positive in psittacosis pneumonia

Prognosis: The mortality rate among old people is said to be between 35 and 40 per cent. Among those under thirty years of age, the mortality is considerably less The convalescence is slow and protracted.

Pneumonia Due to Rickettsial Infection (Q Fever): During 1935[1] there appeared an outbreak of pneumonia among the employees of the National Institute of Health in Washington, D. C. The causative factor recovered from the sputum of the affected persons was later identified as the gram-negative, Rickettsia-like organism responsible for American Q fever (nine-mile fever).

Clinical Course: The onset was acute, with headache, chilly sensations, fever, malaise, coryza, and cough. The sputum was tenacious, mucoid, and white in color.

Physical Signs: Small multiple areas of dullness and few moist râles were revealed. The x-ray examination showed single and multiple areas of soft patchy infiltration resembling those found in atypical virus pneumonia There were fifteen such cases reported, of whom one died

Pulmonary Fibrosis

Pulmonary fibrosis is a chronic condition of the lungs characterized by connective tissue hyperplasia which partially obliterates its air spaces, lymphatics and blood channels. This may result from a variety of factors, such as: (*a*) Chronic inflammatory disease of the lungs, pleura, and bronchi as seen in pulmonary suppuration, chronic pulmonary tuberculosis (chronic fibroid phthisis), pneumonoconiosis, chronic adhesive pleurisy and other chronic affections of the lungs, pleura and bronchi; (*b*) long-standing passive congestion, as seen in

[1] Davis, G E, and Cox, H R.: Pub. Health Rep, 53 - 2259, 1938

interstitial pneumonia is more common than gummata The greatest infiltration is usually found in the pleura and in the connective tissue framework, especially in the interlobar tissue near the root of the lung



Fig 12—Metastatic carcinoma of the lungs (Courtesy of Dr Leon Solis-Cohen.)

Physical Signs: *Inspection* shows diminished expansion, and *palpation* elicits increased tactile fremitus; but if the pleura is involved, diminished tactile fremitus will be elicited. There usually is impaired resonance, and, at times, dullness, over the affected area on *percussion*, while *auscultation* reveals bronchovesicular or bronchial breathing, and in some instances, when an associated effusion, or the plugging of a bronchus occurs, there is an absence of breath sounds

X-ray Findings: There is generally a fan-shaped radiation, extending from the hilum toward the periphery, but not reaching it. This is a general infiltration process and is not confined to the bases alone, though it involves the lower portions of the lungs to a greater extent than the upper. The apices are clear. In *gummata* there are generally discrete shadows of masses in the region of the hilum

Pulmonary Tuberculosis

Pulmonary tuberculosis usually occurs as a chronic, infectious disease of insidious onset and often following a protracted course. The acute types of tuberculosis, the miliary type, may be found in young children and in those who have failed to develop an early immunity.

Symptoms: The general symptoms are those of any chronic wasting disease associated with cough, expectoration, loss of weight and strength, increased temperature, rapid pulse, night sweats, digestive disturbance and hemoptysis The tubercle bacillus is the etiologic factor.

Physical Signs: The physical signs of pulmonary tuberculosis depend upon the stage of the disease, the amount of the involvement, and the rapidity of its progress. The physical signs of the three principal stages of chronic tuberculosis are here considered: *First stage*, or incipient manifest tuberculosis; *second stage*, or moderately advanced tuberculosis and *third stage*, or advanced tuberculosis

First Stage, Incipient Manifest Tuberculosis: ***Symptoms:*** Cough, a slight rise in the evening temperature, exhaustion after slight physical or mental effort, digestive disturbances and neuromuscular pains, dyspnea, particularly on slight exertion and rapid pulse.

Physical Signs: ***Inspection*** may reveal slightly delayed expansion at one or the other apex; *palpation* may elicit

Where the pleura is irritated, a varying amount of fluid is generally present.

Metastatic Malignancy. Metastatic carcinoma in the lung occurs generally in women from a primary focus of the breast, or it may be secondary to carcinoma situated anywhere in the body. Again the hilum shows early involvement and in progressing it may be accompanied by a pleural effusion. Dense infiltration may occur from and along the distribution of the bronchial tree. There is a consequent increase in the bronchial markings and spotting of tumor masses through the lung, or the process may appear simply as areas of light density or rounded, thin plaques, scattered throughout all the lung tissues (See Fig. 12.)



Fig. 11—Tumor of the lungs. (Philadelphia General Hospital.)

The detail simulates that of miliary *tuberculosis.* The areas of involvement, however, are more even and dense than those of the latter.

Syphilis of the Lung

This disease is not readily diagnosed by physical examination alone. A gumma of the lung may give rise to physical signs similar to those elicited over pulmonary tumors, except that the most common location is along the hilum Syphilis may be suspected, if D'Espine's sign is present in conjunction with a positive Wassermann reaction and a previous history of syphilis. These manifestations, together with a tumor along the hilum, make the diagnosis of pulmonary syphilis highly possible. Fibrous

hazy and blurred as a rule because of the amount of exudate in the cells. Along the path of invasion the next change noticed is thickening of the bronchial tissues, extending toward the apex, which is commonly the first area involved. If healing occurs these thickened areas decrease in size, but increase in density, and become sharper in outline. There thickening at this time, particularly in the region of the base. Where the apex and one side is involved we often also find involvement along the vertebral border of the opposite lung It must be remembered that tuberculosis in the adult is usually first observed in the upper portions of the lung. Fluoroscopically the apices are examined for clearness or



Fig 14—Chronic ulcerative phthisis (John C Da Costa, Jr, *Physical Diagnosis*, W. B Saunders Company)

may also be beginning calcification around them Should the invasion and infection continue, other areas of the lung fields become involved and grayish spots (tubercles) appear throughout the parenchymal tissues Fine mottling of the lung tissue is next seen and is always diagnostic of tuberculosis These areas may then fuse and coalesce, and caseation, with or without cavitation, follows. There is more or less interstitial and fibrous density. The patient should be made to cough in order to see if the apices clear up during the act

Miliary Tuberculosis The picture here is different There is fine hazy mottling extending throughout all the lobes. The lung on the x-ray plate gives the appearance of having passed through a snow storm. The apices are always involved, the studding is marked, and the outline of distinctness varies according

slightly increased tactile fremitus, though in some instances where the pleura is thickened diminished tactile fremitus is obtained. Slight rigidity of the muscles is often demonstrable over the affected part. In the very early stages no changes in the *percussion* note are appreciable When sufficient infiltration has occurred,



Fig 13—Pulmonary tuberculosis (Courtesy Dr. Leon Solis-Cohen)

the percussion note is usually impaired and the pitch somewhat elevated. *Auscultation* may reveal a somewhat lengthened expiratory note, while the inspiratory sound is a trifle harsher than normal. When activity is present, fine moist râles are demonstrable after cough. In open cases tubercle bacilli will be found in the sputum

Second Stage, Moderately Advanced: ***Symptoms:*** Cough, with or without expectoration and at times hemoptysis, rapid loss of weight and strength, evening rise of temperature, night sweats, digestive disturbances, nervous irritability and exhaustion

Physical Signs: ***Inspection*** shows delayed and often diminished expansion; *palpation* confirms inspection, and elicits increased tactile fremitus; *percussion* yields relative dullness, and *auscultation* reveals bronchovesicular breathing. increased vocal resonance, subcrepitant and small bubbling râles, particularly after cough. Tubercle bacilli in the sputum, and x-ray evidence of the disease is common at this stage

Third Stage, Advanced Tuberculosis: ***Symptoms:*** Emaciation, cough expectoration, elevated temperature, asthenia and night sweats.

Physical Signs: Retraction of the affected parts is shown by *inspection,* together with delayed and diminished expansion; *palpation* confirms inspection, and elicits increased tactile fremitus. *Percussion* over consolidated areas yields dullness; over a cavity, tympany *Auscultation* over consolidated areas reveals bronchial breathing, bronchophony, and small moist râles; over a cavity, cavernous or amphoric breathing, whispered pectoriloquy and moist râles Associated pleural effusion or pneumothorax will materially alter the physical signs.

X-ray Findings: *Pulmonary Tuberculosis:* Advanced tuberculosis is not difficult of detection It is the early case which calls for the greater care. A careful comparison of the clinical report and the case history with the x-ray findings is necessary. (See Fig 13.)

Any increase and thickening of the bronchial markings must be viewed with suspicion Fine mottling along the bronchi is generally due to early tuberculosis. This is accompanied by exaggeration of the root shadows In the acute stage the area involved is not as distinct as the adjacent areas, and it is

Classification of Pulmonary Tuberculosis (*Continued*)

LESION	POSSIBLE SYMPTOMS
III *Far Advanced Lesion:* A lesion more extensive than under *moderately advanced* Or definite evidence or marked cavity formation. Or serious tuberculous complications.	*A. Symptoms Slight or None* (Same as *A* above). *B. Symptoms Moderate:* (Same as *B* above). *C. Symptoms Severe* (Same as *C* above).

Under this scheme the following classifications are possible.

Minimal A	Moderately Advanced A	Far Advanced A	
" B	" " B	" " B	
" C	" " C	" " C	

Apparently Cured

All constitutional symptoms and expectoration with bacilli absent for a period of two years under ordinary conditions of life

Arrested:

All constitutional symptoms and expectoration with bacilli absent for a period of six months; the physical signs to be those of a healed lesion; roentgen findings to be compatible with the physical signs.

Apparently Arrested·

All constitutional symptoms and expectoration with bacilli absent for a period of three months, the physical signs to be those of a healed lesion; roentgen findings to be compatible with physical signs

Quiescent·

Absence of all constitutional symptoms; expectoration and bacilli may or may not be present, physical signs and roentgen findings to be those of a stationary or retrogressive lesion; the foregoing conditions to have existed for at least two months.

Improved

Constitutional symptoms lessened or entirely absent; cough and expectoration with bacilli usually present; physical signs and roentgen findings to be those of a stationary or retrogressive lesion.

Unimproved

Essential symptoms unabated or increased; physical signs and roentgen findings to be those of an active or progressive lesion.

found weakness, rapid pulse, irregularly appearing sweats, and signs of disseminated bronchitis, with one or more concentrated areas which yield bronchopneumonic signs In the absence of tubercle bacilli in the sputum, an x-ray examination will usually reveal the true nature of the disease.

Acute Pneumonic Phthisis

This is an acute infiltration of the lungs, pneumonia-like in character, the specific organisms of which are chiefly the tubercle bacilli.

Symptoms: The symptoms are acute onset, high fever, frequent sweats with attacks of chilliness, high temperature, either of the pneumonic or septic type The temperature curve depends upon the kind of bacteria growing in symbiosis with the tubercle bacilli.

Physical Signs: The physical signs are those of lobar and at times of lobular

to the stage of involvement. *Malignancy* and *pneumonoconiosis* may often cast similar shadows, but in malignancy there are not so many areas of greater density and sharper detail, while in pneumonoconiosis the condition is widespread, but does not involve the apices, the diseased areas being smaller and distinct in outline without involvement of the surrounding tissue. (Fig. 15.)

The following classifications of pulmonary tuberculosis as to the stage of the disease and the state of repair are adopted at the Sanatorium for Consumptives, Eagleville, Pa.

Acute Miliary Tuberculosis

Acute miliary tuberculosis is characterized by its acute onset, high fever and the formation of miliary tubercules in the lungs and in nearly all the tissues of the body.

Symptoms: These may simulate those of typhoid fever, bacterial endocarditis, septicema, peribronchial and abdominal Hodgkin's disease, etc.

Physical Signs: In the lungs these are often misleading, though the presence of the disease may be suspected by the somewhat irregular, though continuous, elevation of temperature, pro-

Classification of Pulmonary Tuberculosis

Lesion	Possible Symptoms
I. *Minimal Lesion:* Slight lesion or lesions limited in total volume to that above the second chondrosternal junction and fifth thoracic vertebra of one side. No serious tuberculous complications	A *Slight or None* Slight or no constitutional symptoms including (particularly) gastric or intestinal disturbance or rapid loss of weight; slight or no elevation of temperature (not over 99.5° F. after rest) or acceleration of pulse (not over 90 for men and 96 per minute for women after rest) at any time during the 24 hours. Expectoration usually small in amount or absent Tubercle bacilli may be present or absent.
	B. *Moderate* No marked impairment of function, either local or constitutional
	C *Severe* Marked impairment of function, local or constitutional
II: *Moderately Advanced Lesion:* A lesion of one or both lungs, more widely distributed than under *minimal*, the extent of which may vary, according to the severity of the disease, from the equivalent of one-third the volume of one lung (consolidation or marked infiltration) to the equivalent of the volume of an entire lung (infiltration) with or without evidence of cavity formation, cavities not to exceed in total diameters 2 cm. No serious tuberculous complications.	A. *Symptoms Slight or None* (Same as *A* above).
	B *Symptoms Moderate* (Same as *B* above)
	C *Symptoms Severe:* (Same as *C* above).

Classification of Pulmonary Tuberculosis (*Continued*)

Lesion	Possible Symptoms
III *Far Advanced Lesion* A lesion more extensive than under *moderately advanced.* Or definite evidence or marked cavity formation Or serious tuberculous complications	*A Symptoms Slight or None:* (Same as *A* above). *B Symptoms Moderate·* (Same as *B* above). *C. Symptoms Severe·* (Same as *C* above).

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" C	" " C	" " C

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Absence of all constitutional symptoms, expectoration and bacilli may or may not be present; physical signs and roentgen findings to be those of a stationary or retrogressive lesion; the foregoing conditions to have existed for at least two months

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Constitutional symptoms lessened or entirely absent; cough and expectoration with bacilli usually present; physical signs and roentgen findings to be those of a stationary or retrogressive lesion

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Essential symptoms unabated or increased; physical signs and roentgen findings to be those of an active or progressive lesion

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Acute Pneumonic Phthisis

This is an acute infiltration of the lungs, pneumonia-like in character, the specific organisms of which are chiefly the tubercle bacilli.

Symptoms: The symptoms are acute onset, high fever, frequent sweats with attacks of chilliness, high temperature, either of the pneumonic or septic type The temperature curve depends upon the kind of bacteria growing in symbiosis with the tubercle bacilli

Physical Signs: The physical signs are those of lobar and at times of lobular

person during an allergic seizure and in one suffering from parasitic infestations, or in one who has marked eosinophilia from any cause.

Mycotic (Fungus) Infections of the Lungs

General Consideration: The fungi belong to the plant kingdom and are classified as the Thallophyta, that is simple filamentous structures devoid of roots, stems, and leaves. There are two types of Thallophyta: (1) the *algae*, containing chlorophyll, which allows them to manufacture their own food, and (2) the *fungi*, which are devoid of chlorophyll, and are therefore parasitic or saprophytic.

The fungi are usually divided into two large subgroups: the *Pseudomycetes*, or false fungi, and the *Eumycetes*, or true fungi. *Pseudomycetes* are further divided into two classes: the *Schizomycetes*, or bacteria, to which class belong the human pathogenic fungi, *Actinomycetes* and *Nocardia*, and *Myxomycetes*, or slime mold, which are nonpathogenic for humans.

The Eumycetes, or true fungi, are subdivided into four classes, the *Phycomycetes*, the *Ascomycetes*, the *Basidiomycetes*, and the *Fungi Imperfecti*

Fungi Imperfecti: To this group belong the majority of organisms which are pathogenic for man. They lack a sexual stage in their life cycle and are characterized by a vegetative body, composed of filamentous elements, or hyphae, and reproductive organs, or various types of spores, which are produced in a number of ways

The mycoses are of two types: the *superficial* which affect the skin, and the systemic which affect the viscera.

The pulmonary mycoses are comparatively rare. The various fungi that infect the respiratory tract cause lesions that are at times indistinguishable from various stages of pulmonary tuberculosis The fungi may reach the bronchopulmonary system directly by inhalation of their spores, through the blood and lymph channels, or by extension from contiguous structures.

ACTINOMYCOSIS (STREPTOTHRICOSIS, NOCARDOSIS, LUMPY JAW)

Actinomycosis is a chronic, suppurating, granulomatous infection caused by various *species of Actinomyces* or Nocardia. This is a group of fungi closely related to bacteria. The disease is characterized by the formation of multiple abscesses and fistulae, in the drainage of which are found the characteristic granules of the fungus.

Pulmonary Actinomycosis: Primary infection of the lungs may result from aspiration of infected material from the mouth, or it may be inhaled with dust, straw, grain, weeds, soil, or other infected material Occasionally the infection may reach the lungs from outside the respiratory tract through sinuses in the chest wall

Clinical Course: The onset is gradual. During the first few weeks of the disease, there is a low irregular temperature, cough, and some expectoration. As the disease progresses, the symptoms become intensified The cough is more troublesome, and the sputum becomes mucopurulent and may contain blood. Small abscesses develop in the lung. The infection may extend to the pleurae, the mediastinum, the pericardium, and the heart, and numerous discharging sinuses may form in the chest wall When suppuration occurs, the temperature becomes spiked, respirations become labored, and night sweats develop The patient loses

weight and strength and becomes anemic. Dysphagia develops when the mediastinum becomes involved

Physical Signs: During the early stages, the physical signs resemble those of basal pulmonary tuberculosis, since the lesions generally occur at the bases As the disease progresses, there develop massive lesions in one or both lungs.

X-ray Findings: The roentgenologic picture is that of massive consolidation, often containing small, cavity-like, rarefied areas

Diagnosis: The diagnosis may be made on finding the sulfur granules in the sputum or in material discharged from the chest sinuses, or by finding characteristic lesions elsewhere in the body

Prognosis: This disease runs a chronic course and usually terminates fatally

COCCIDIOIDOMYCOSIS (SAN JOAQUIN FEVER, VALLEY FEVER, DESERT RHEUMATISM, POSADA-WERNICKE'S DISEASE)

Pulmonary coccidioidomycosis is a highly infectious disease caused by Coccidioides immitis It occurs in two forms (1) primary coccidioidomycosis which is usually a benign, self-limiting, respiratory disease, and (2) progressive coccidioidomycosis which is a chronic, malignant, and disseminated disease involving *also the cutaneous, subcutaneous,* visceral, and osseous tissue

This disease occurs endemically in the San Joaquin Valley in California; it is also found in Arizona, New Mexico, Texas, and other arid areas of the southwestern part of the United States It may also be found sporadically in warm climates elsewhere

Etiology: Coccicioides immitis is a fungus pathogenic to animal and man It is believed that coccidioidomycosis is primarily a disease of rodents in whom it occurs endemically. Infection in man is caused by the inhalation of dust containing the chlamydospores It appears that the disease is not transmitted by contact directly from animals to man or from man to man.

Clinical Course: The incubation period is from ten to fourteen days The onset is gradual with low-grade fever (99° to 101° F), mild cough with very little expectoration, and a moderate degree of nasopharyngitis Mild cases may attract little attention. The moderately severe cases may develop chills, night sweats, headache, backache, and anorexia. The cough becomes troublesome, and small amounts of blood-streaked mucopurulent material are raised Dry pleurisy and, in rare instances, pleurisy with effusion are among the early manifestations

Physical Examination: In the absence of pleurisy, the physical findings in the lungs are meager. Skin lesions resembling erythema nodosum may appear over the scalp, arms, buttocks, thighs, and shins in a small number of cases Other lesions resembling erythema multiforme may appear on the margins of the palms, face, neck, and upper extremities These lesions are of an allergic nature Occasionally acute arthritis may develop, affecting chiefly the knees and ankles

X-ray Examination of the Lungs: This may show either isolated circumscribed lesions in the bases of the lungs, or the lesions may be multiple, resembling metastatic foci or primary nodular tuberculosis The lung lesions are benign and may resolve within a few months or develop into thin-walled cavities which eventually shrink and become calcified Other types of lesions may show roentgenographically only as fuzzy hilar thickenings or as hazy areas of infiltration extending from the hilum towards the

periphery in the lower lung fields. These lesions may resolve within a few weeks or they may persist for several months after the clinical symptoms have subsided.

Blood Count: During the early stages, there is a moderate leukocytosis with a normal differential count. Later there develops a lymphocytosis with increased mononucleosis.

Prognosis: The majority of patients recover within two or three weeks; complications are usually absent The disease usually responds to the intramuscular injections of colloidal copper

Diagnosis: The diagnosis is based upon (1) the demonstration of the nonbudding, thick-walled spores containing numerous endospores; (2) the development of lesions containing the typical spore, obtained by intratesticular injections of guinea pigs; and (3) by obtaining a positive complement-fixation reaction.

Progressive Coccidioidomycosis: This runs a severe and progressive course often terminating fatally All the symptoms of the disease are intensified. The lung lesions are prominent and resemble tuberculosis, and there is often invasion of the bony structures. Mediastinal adenopathy is a frequent accompaniment of the progressive type of this disease

Moniliasis *(see page 1095)*

Moniliasis is caused by a yeast-like fungus, Candida albicans It occurs as an acute or subacute infection invading the skin, nails, vagina, bronchi, and lungs.

Bronchopulmonary Moniliasis (Bronchomycosis): This occurs in two forms: bronchial moniliasis and pulmonary moniliasis

Bronchial Moniliasis: This is more common than the pulmonary type. A particular form of this infection has been described among tea-tasters of Ceylon. The symptoms are not characteristic; they resemble bronchitis.

Symptoms: There are no specific symptoms except cough and expectoration. The health is usually unimpaired. The cough, however, is distressing. The expectoration is copious, almost colorless, and is of mucoid and gelatinous consistency, containing small gray flakes composed of budding fungous cells and other material.

Physical Examination: This usually reveals medium-sized and coarse râles at the bases.

X-ray Findings: Roentgenologically the picture is nonspecific. There is some peribronchial thickening with a hazy type of linear fibrosis.

Diagnosis This is made by discovering the Candida (Monilia) albicans in the sputum.

Prognosis. The infection may disappear spontaneously, or it may drag along for years with periodic remissions and exacerbations.

Pulmonary Moniliasis: This form is less common but more severe than the bronchial type. At the onset there is a rise in temperature and cough. The temperature may range between 100° and 103° F., it is irregular and often accompanied by sweats. The cough is severe, and the sputum is mucoid and gelatinous, often blood-streaked. Pleural pain and, at times, effusion may occur at the onset or may develop later in the disease.

Physical Examination: The lungs show patchy consolidations distributed in various areas resembling bronchopneumonia or atypical pneumonia. Occasionally the lobular consolidation may become confluent and give the picture of lobar pneumonia

X-ray Findings: The x-ray findings usually show ill-defined areas of consolidation in one or both lungs. These lesions are migratory, they do not affect the apices. Occasionally an entire lobe or more than one lobe may show massive consolidation. In the presence of pleural effusion, the x-ray picture is characteristic.

Laboratory Tests: The leukocyte count is normal, but the neutrophiles are moderately increased. The sedimentation rate is normal.

Diagnosis. This depends largely on finding the fungus in the sputum. The skin test with Candida albicans is positive, and agglutinins in the blood may give a positive reaction in dilutions of from 1:80 to 1:2400. Occasionally no reaction appears. The fungus may be cultured in Sabouraud's glucose medium.

Prognosis: Some patients may recover spontaneously within a few weeks, others may develop chronic bronchiolitis. Complete consolidation of two or more lobes usually terminates fatally.

Histoplasmosis of Darling

Histoplasmosis is caused by Histoplasma capsulatum. This organism is primarily a parasite of the reticuloendothelial system resembling Leishmania donovani, but it is lacking in blepharoplast. The fungus may appear in two forms: one, yeast-like when recovered from the reticuloendothelial tissue and the blood; the other, a mycelial form when cultured outside the body. Histoplasmosis is more common in children than in adults. It is a chronic disease characterized by the occurrence of fever, emaciation, anemia, leukopenia, splenomegaly, and lesions on the skin, of the bones, and in the lungs.

Pulmonary histoplasmosis is characterized by the formation of multiple, small, noncaseating tubercles, consisting of infected macrophages and distributed in the alveolar spaces and interstitial tissues.

Symptoms: The pulmonary symptoms are pleural pain, cough, and occasional hemoptysis. The spleen and lymph nodes are enlarged, resembling Hodgkin's disease, leukemia, or lymphosarcoma.

X-ray Findings: X-ray examination may show some enlarged hilar lymph nodes with some peribronchial thickening.

Blood Count: This usually shows signs of severe secondary anemia and either a mild leukocytosis or a marked leukopenia.

Diagnosis: This is made on recovery of the fungus from the blood and bone-marrow smears, and from material obtained from splenic puncture or biopsied lymph gland, or from cultures made of guinea pigs or mice infected with infected material. The disease may accompany other diseases and is particularly prone to accompany tuberculosis.

Histoplasmosis has been reported from Central America, South America, the United States, England, the Philippines, and Java. The organism is believed to enter the body by way of the mouth or the intestinal tract.

Prognosis: Histoplasmosis is a fatal disease, children usually succumb to the disease within a few weeks. Adults may live from three to eight months. Occasionally one may live for several years after contracting the disease, the period being marked with remissions and exacerbations.

Blastomycosis (Gilchrist's Disease)

(see page 1094)

Blastomycosis is a chronic, granulomatous, and suppurative process, caused by

the Blastomyces dermatitidis, which appears in the tissues as budding, round, or ovoid cells, and in culture, at room temperature, as a filamentous, mold-like organism.

Two types of the disease are described: (1) North American blastomycosis (Gilchrist's disease); and (2) South American blastomycosis (Lutz-Splendore-Almeida's disease).

There are two types of North American blastomycosis. (1) the cutaneous type, which is characterized by the formation of granulomatous, ulcerative lesions upon any skin surface of the body; and (2) the visceral, or systemic type, which affects chiefly the lungs, bony structures, the abdominal viscera, and the nervous system

Pulmonary Blastomycosis: The infection reaches the lungs chiefly by inhalation of the infected material.

Clinical Course: The onset is usually insidious, as a subacute respiratory infection There is a low-grade temperature, some dyspnea, a dry hacking cough, and some chest pain As the disease progresses, the symptoms become intensified The temperature reaches a higher level, the cough becomes troublesome, and there is purulent and often blood-streaked sputum. Dyspnea, weakness, emaciation, and night sweats become troublesome, and lesions may develop upon the skin or internal viscera

Physical Signs: The physical findings in the lungs suggest either massive pulmonary tuberculosis, pulmonary abscess, or neoplasm Other findings, such as subcutaneous abscesses with discharging sinuses, may be found on the chest or other parts of the body.

X-ray Examination: During the early stages, the x-ray plates may show mild parenchymal infiltration with enlarged mediastinal lymph nodes. As the disease progresses, the x-ray film will show irregular dense masses projecting from the hilum, which resemble neoplasm. Later in the disease, the roentgenographic picture is that of a unilateral or bilateral dense pulmonary lesion containing small cavities. The ribs, spinal vertebrae, or other bones may show x-ray evidence of a destructive process.

Laboratory Findings: The blood count reveals a secondary anemia and leukocytosis with an increase in the neutrophiles. Microscopic examination of the sputum, of the pus from a discharging focus, or of the scrapings from a cutaneous lesion may show the fungus, or the fungus may be revealed by culturing these materials.

Diagnosis: This is based on the history, clinical and x-ray findings, and the presence of the fungus by microscopic examination or by culture.

Prognosis: The disease is chronic; in the pulmonary form, death usually occurs within two years

Geotrichosis

Geotrichosis is a rare disease, often resembling North American blastomycosis It is caused by a species of Geotrichum, a fungus sometimes found in the mouth and intestines of normal persons Like moniliasis, it may be localized within the bronchial tree, or it may affect the lungs proper.

Bronchial Geotrichosis: *Clinical Features:* The clinical features of bronchial geotrichosis resemble bronchitis caused by any other organism The temperature is usually normal, and there are few systemic manifestations. The symptoms and physical signs are persistent cough and the expectoration of blood-streaked, gelatinous, or mucoid sputum.

X-ray Findings: Roentgenological examination will show diffuse peribronchial thickening, with some areas of mottling at the bases

Pulmonary Geotrichosis: This type of infection is more severe than the bronchial form. The symptoms and physical signs resemble pulmonary tuberculosis

Clinical Course: The invasion of the lungs by the fungus is marked by a rise in temperature which continues throughout the disease. The general symptoms are headache, some night sweats, and cough with light-colored, mucopurulent expectoration which contains the fungus Occasionally the sputum is blood-streaked, or there may be hemoptysis

Physical Signs: The physical findings resemble those of pulmonary tuberculosis and depend upon the type of pulmonary lesion present at the time of the examination

X-ray Examination: This may reveal various types of lesions depending upon the stage of the infection The lesions may occur in any part of the lungs, though usually they are located in the upper parts of the lungs They consist of dense patches of consolidation Thin-walled cavities may be found either within the consolidated areas or adjacent to them

Laboratory Examination: The blood count and urinalysis do not reveal anything abnormal Microscopic examination of the sputum will reveal the Geotrichum The fungus can also be discovered by culturing the sputum and feces. These organisms may at times be found in conjunction with the Friedlander's bacilli or the Mycobacterium tuberculosis in the sputum of those suffering from Friedländer's bacillus infection and from pulmonary tuberculosis

Diagnosis: Since the Geotrichum is at times found in the secretions of normal persons, and since it may be a secondary invader in tuberculosis, Friedlander's pneumonitis, and other infections, a positive diagnosis of pulmonary geotrichosis should be made only when the Geotrichum is persistently found in large numbers in the bronchial secretions.

Prognosis: Nearly all the reported cases of this disease have recovered.

Cryptococcosis (Torulosis, European Blastomycosis, Busse-Buschke's Disease)

Cryptococcosis is a subacute or chronic infection caused by Cryptococcus neoformans (Torula histolytica) In the tissue it appears as an ovoid or spherical, single-budding, thick-walled, yeast-like fungus surrounded by a refractile gelatinous capsule. It may involve any part of the body, and is found in the skin, the lungs, the brain, and meninges

Pulmonary cryptococcosis (torulosis) is not readily diagnosed, since symptoms and physical signs are not specific.

Clinical Course: There is a low-grade fever with a mild hacking cough The expectoration is scanty, mucoid in character, and is occasionally blood-streaked

Physical Signs: They are meager in proportion to the x-ray findings Over larger consolidations, there is impaired resonance and bronchovesicular breath sounds Râles are found only in the presence of widely disseminated lesions.

X-ray Findings: Roentgenologic examination may show dense massive lesions resembling pulmonary tuberculosis, neoplasm, or resolving pneumonitis

Laboratory Examination: The blood count may show a secondary anemia, the leukocyte count may be normal or some-

the Blastomyces dermatitidis, which appears in the tissues as budding, round, or ovoid cells, and in culture, at room temperature, as a filamentous, mold-like organism.

Two types of the disease are described: (1) North American blastomycosis (Gilchrist's disease); and (2) South American blastomycosis (Lutz-Splendore-Almeida's disease).

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Pulmonary Blastomycosis: The infection reaches the lungs chiefly by inhalation of the infected material

Clinical Course: The onset is usually insidious, as a subacute respiratory infection There is a low-grade temperature, some dyspnea, a dry hacking cough, and some chest pain As the disease progresses, the symptoms become intensified The temperature reaches a higher level, the cough becomes troublesome, and there is purulent and often blood-streaked sputum. Dyspnea, weakness, emaciation, and night sweats become troublesome, and lesions may develop upon the skin or internal viscera

Physical Signs: The physical findings in the lungs suggest either massive pulmonary tuberculosis, pulmonary abscess, or neoplasm. Other findings, such as subcutaneous abscesses with discharging sinuses, may be found on the chest or other parts of the body.

X-ray Examination: During the early stages, the x-ray plates may show mild parenchymal infiltration with enlarged mediastinal lymph nodes. As the disease progresses, the x-ray film will show irregular dense masses projecting from the hilum, which resemble neoplasm. Later in the disease, the roentgenographic picture is that of a unilateral or bilateral dense pulmonary lesion containing small cavities. The ribs, spinal vertebrae, or other bones may show x-ray evidence of a destructive process.

Laboratory Findings: The blood count reveals a secondary anemia and leukocytosis with an increase in the neutrophiles Microscopic examination of the sputum, of the pus from a discharging focus, or of the scrapings from a cutaneous lesion may show the fungus, or the fungus may be revealed by culturing these materials

Diagnosis: This is based on the history, clinical and x-ray findings, and the presence of the fungus by microscopic examination or by culture

Prognosis: The disease is chronic; in the pulmonary form, death usually occurs within two years

Geotrichosis

Geotrichosis is a rare disease, often resembling North American blastomycosis It is caused by a species of Geotrichum, a fungus sometimes found in the mouth and intestines of normal persons Like moniliasis, it may be localized within the bronchial tree, or it may affect the lungs proper.

Bronchial Geotrichosis: *Clinical Features:* The clinical features of bronchial geotrichosis resemble bronchitis caused by any other organism. The temperature is usually normal, and there are few systemic manifestations. The symptoms and physical signs are persistent cough and the expectoration of blood-streaked, gelatinous, or mucoid sputum

and lung abscess may be severe and often prove fatal Hemoptysis occurs frequently in cysts communicating with bronchi. The clotted blood within the cyst retracts and has a characteristic radiologic appearance It is likened to the appearance of "a stone loose within its setting"

Dermoid Cysts of the Lung: Dermoid cysts may occur in most structures of the body When they occur in the lungs, their clinical manifestations depend upon the size of the cyst and its location Small cysts, when they do not obstruct the bronchus, may be asymptomatic Large cysts may cause pressure symptoms, such as dyspnea, cough, and signs of pulmonary congestion The physical signs depend upon the location and the size of the mass At the periphery, the signs are those of tumor Roentgenologic examination will discover the position of the mass, and the finding of teeth and other radiolucent substances

Acquired Cysts of the Lungs: Various types and sizes of cysts may develop within the lung as a result of general infection with localized foci in the lungs

Echinococcus Cysts (Hydatid Disease of the Lungs): The embryos of the dog tapeworm reach the lungs, liver, and other structures by way of the blood and lymph circulation The cysts in the lungs are unilocular, and often solitary

Diagnosis: The patient may remain asymptomatic as long as the cyst is small When it reaches a certain size, particularly when close to a bronchus, it will cause cough, dyspnea, and, at times, hemoptysis The physical signs may be those of a solid tumor A blood count will reveal a moderate, to a marked, eosinophilia When the cyst is ruptured, anaphylactic shock may ensue or the patient will drown in the cystic fluid. The fluid may contain hooklets and scolices. Radiologic examination before the cyst ruptures will reveal a round or oval shadow with a clear cut edge After the cyst has ruptured, if within a bronchus, an air cyst will be seen, and if it ruptures into the pleural cavity, the picture will be that of a hydrothorax.

Other cysts in the lungs may form as a result of irritation by the inhalation of gases, foreign bodies, or other irritants These may be asymptomatic or they may cause bronchiectasis, pulmonary fibrosis, or pulmonary suppuration.

Sarcoidosis (Besnier-Boeck-Schaumann Disease)

This is a chronic benign granulomatosis of unknown etiology. It may occur in almost any part of the body, but has a predilection for lymphoid tissue, the uveal tract, iris, and cornea The disease usually begins in childhood or early adulthood The lesions consists of hard, rubbery, discrete tubercles, made up of a miliary collection of large pale-staining epithelioid cells, concentrically arranged, without central necrosis Occasionally there are irregular giant cells whose many nuclei are pale and contain peculiar inclusions.

Pulmonary Sarcoidosis: The lesions in the lungs resemble those of miliary tuberculosis or Hodgkin's disease

Four types of the disease are seen. (1) bilateral involvement of hilar nodes, (2) miliary, nodular, bilateral involvement which later undergoes spontaneous regression, (3) soft infiltration similar to caseous tuberculosis, and (4) dense hilar radiation into the median and lower lobes (F K. Albrecht).

what elevated. The sedimentation rate is increased. The spinal fluid pressure is increased, it is xanthochromic or turbid. The cell count is variable, 200 to 800 cells per cu. mm. The protein content is increased. Glucose may be normal or somewhat decreased. The colloidal gold curve resembles meningitis.

Diagnosis: The diagnosis is made microscopically by finding the fungus in the sputum and spinal fluid, by culture, by animal inoculation, and by biopsy of the skin or lymph node.

Prognosis. Uncomplicated pulmonary cryptococcosis may recover. In the presence of meningeal involvement, which is common, the prognosis is grave.

Aspergillosis

Aspergillosis is an inflammatory granulomatous infection occurring in the skin, external ear, nasal sinuses, bronchi, lungs, bones, and meninges. It is caused by certain species of Aspergillus, the most common fungous contaminant in the laboratory.

Pulmonary Aspergillosis: This condition is rarely diagnosed ante mortem. The symptoms and physical signs resemble those found in pulmonary tuberculosis. There is remittent fever, loss of weight, and cough with bloodstained, mucopurulent expectoration.

X-ray Examination: This will show a picture of either diffuse nodular lesions or smooth dense lesions, with or without cavitation in various parts of the lungs.

Diagnosis: The constant presence of the branching hyphae in the sputum of patients who present tuberculous-like lesions in the lungs is suspicious of pulmonary aspergillosis.

Prognosis: Mild cases, in whom the lesions are limited to the bronchi, recover. In the presence of massive pulmonary consolidation with abscess formation, the prognosis is grave.

Cysts of the Lungs

Cysts of the lungs may be congenital or acquired.

Congenital Cystic Disease of the Lungs: This is a comparatively rare condition. The cysts may be in the bronchi or in the lung proper. They may be solitary or multiple, large or small, communicating or noncommunicating with the bronchi. Congenital bronchial cysts, which do not communicate with bronchi, contain fluid. They are usually asymptomatic unless they become secondarily infected or rupture into a bronchus. Cysts that do communicate with a bronchus contain air and cause few characteristic symptoms. In the presence of a narrow portion of the lumen of a bronchus with redundancy of its mucosa, a check-valve action takes place, which gradually entraps the inspired air, thus increasing the size of the cyst and causing great intrapulmonary tension, resembling pneumothorax.

Diagnosis: These cysts, when uncomplicated, are often asymptomatic and are only discovered by x-ray examination. When the cysts are large, there is dyspnea on exertion, and signs of pneumothorax. The x-ray examination will show the presence of lung tissue at the hilum and traces of pulmonary tissue at the periphery on the affected side. This differs from the x-ray findings in pneumothorax where such lung tissue is not seen. Multiple small cysts have a soap-bubble appearance on a roentgenogram. Lipiodol injection will show the position of cysts when they communicate with bronchi.

Complications: Secondary infection such as bronchitis, septic pneumonia

or relative dullness, the difference being caused by the thickness of the pleura, that is, the thicker the pleura, the duller the percussion note. Distant breath sounds are heard on *auscultation*.

Serofibrinous Pleurisy (pleurisy with effusion, subacute pleurisy): Serofibrinous pleurisy may be the second stage of fibrinous pleurisy. Its most common cause is pulmonary tuberculosis, at least 85 to 90 per cent of all cases of serofibrinous pleurisy being attributable to this condition. Lobar pneumonia and acute articular rheumatism are the next most common causes.

Symptoms: These are shortness of breath, elevation of temperature, and mild toxic symptoms.

Physical Signs: Over small effusions *inspection* shows diminished expansion, over large effusions there will be absence of expansion. *Palpation* elicits weak tactile fremitus over small effusions; none over large effusions. There will be relative dullness on *percussion* over small effusions; over large effusions, flatness and positive Grocco's sign. *Auscultation* over small effusions reveals distant breath sounds; over large effusions absence of breath sounds is the rule. If the effusion is not bound down by adhesions, a change of posture will bring about a gradual alteration in the upper line of dullness.

Pleural Effusions

Effusions in the pleura may be of several types as follows: (*a*) Hydrothorax (serum); (*b*) pyothorax or empyema (pus); (*c*) hemothorax (blood); (*d*) chylothorax (lymph); (*e*) pneumothorax (air); (*f*) hydropneumothorax (serum and air). and (*g*) pyopneumothorax (pus and air).

Hydrothorax: *Physical Signs:* The signs of hydrothorax are similar to those of pleural effusions, the difference being that in hydrothorax there is no elevation of temperature; an exploratory puncture will reveal fluid of a noninflammatory character (transudate). Hydrothorax usually occurs as a result of heart failure and general dropsy due to either a pathologic kidney condition or severe anemia and also to neoplasm of the lung or pleura.

Pyothorax: If the pus is not bound down by adhesions, the physical signs are similar to those of pleural effusions.

Symptoms: They are of a septic infection; chills, fever, sweating and irregular temperature.

Physical Signs: A localized collection of pus in the pleura (emphyema) may be discovered by its strict localization; *palpation* will elicit absence of tactile fremitus. There will be localized dullness on *percussion* and absence of breath sounds on *auscultation* over the affected area. Exploratory puncture will reveal pus.

Hemothorax: This signifies blood in the pleural cavity. It may be of two types: The first, in which the effusion is free blood, is usually the result of a ruptured blood vessel, of an aneurysm or of trauma. The second, in which blood is so mixed with fluid that the effusion is only bloodstained, may be found in neoplasm of the lung and at times in pulmonary tuberculosis. The symptoms and physical signs are similar to afebrile pleurisy with effusion. Thoracentesis is of diagnostic importance.

Chylothorax: *Symptoms* and *physical signs* are those of pleurisy with effusion, the diagnosis being made by exploratory puncture.

Symptoms: The patient experiences little discomfort The *temperature is* usually normal; the pulse rate and respiratory rate are unaffected There *may be some malaise*, poor appetite, vague gastric symptoms, and diarrhea.

Physical Signs: Extensive intrathoracic involvement may occur in some cases without any definite subjective or objective manifestations In others, there may be evidence of an intrathoracic mass with pressure symptoms, such as cough, dyspnea, some cardiac embarrassment, and a remittent, low-grade fever

Laboratory Findings: There may be a slight secondary anemia, normal or slight decrease in leukocytes, increase in monocytes, and slight eosinophilia. The plasma proteins are increased, and the albumin globulin ratio is often reversed The sedimentation rate is rapid.

Roentgenographic Examination: This may show involvement of the mediastinal lymph nodes, or one or more large masses in the lungs The picture may also be that of miliary tuberculosis, Hodgkin's disease, or malignancy Phalanges or other bones usually show small, circumscribed punched-out areas.

Diagnosis: Uncomplicated pulmonary sarcoidosis is rare; when it does occur, the disease may be inferred from *mild pulmonary symptoms* with definite x-ray findings There is a rapid sedimentation rate and increased serum *globulin. However, the great majority* of patients with pulmonary sarcoidosis exhibit evidence of the disease elsewhere *in the body The cervical* and parotid glands may be enlarged, the face and eyes may show evidence of invasion, and the *hands may* exhibit firm, symmetrically distributed nodules at the interphalangeal joints There may also be neurologic involvement. A biopsy of an accessible gland will aid in differentiating *sarcoidosis from tuberculosis*, Hodgkin's disease, and malignancy. A Wassermann test will exclude syphilis.

Diseases of the Pleura

Pleurisy

Plastic pleurisy (dry fibrinous pleurisy) may be either acute or chronic.

Acute Plastic Pleurisy: This condition may be caused by exposure to wet and cold; by bacterial invasion or by internal or external injury; it is also found in connection with such diseases of the lungs as lobar pneumonia. pulmonary tuberculosis, etc. Chronic pleurisy is found in chronic lung diseases such as tuberculosis, fibroid induration of the lung, syphilis and malignant diseases.

Symptoms: The most prominent symptom of dry pleurisy is a "stitch-like" pain in the side on respiration.

Physical Signs: ***Inspection*** shows that the patient has a tendency to lean toward and favor the affected side, thereby voluntarily inhibiting the respiratory expansion of that side Friction fremitus may be ***palpated*** over the affected area, ***percussion*** is usually negative before the formation of an exudate. On ***auscultation*** a friction rub is heard at the site of the inflammation before the appearance of an exudate and also after its partial absorption

Chronic Plastic Pleurisy: This form may be diagnosed by the history and the following ***physical signs:*** Diminished expansion over the affected side is shown by ***inspection. Palpation*** elicits decreased tactile fremitus, except when fibrous bands stretching from a bronchus to the pleura are formed, in which case, increased tactile fremitus is elicited ***Percussion*** yields impaired resonance

and by the lung border. A partial or complete collapse of the lung may occur, according to the extent of the pneumothorax. Fluid is sometimes present as hydro- or pyopneumothorax and this will be indicated by the density of the fluid shadow beneath the transparent air

Encysted Fluid: This will show a cavity with a fluid level and a smooth, circumscribed wall. It appears to be of uniform density and air may overlie the fluid. It is quite often difficult to differentiate between an encysted empyema and a collection of pus in the intralobar areas, as both are found in contact with the pleura.

Diseases of the Diaphragm

The diaphragm is normally one of the most active muscles in the body, as it constantly contracts and relaxes in its effort to carry on respiration There are certain pathological conditions which affect the diaphragm, thereby interfering with its proper function.

Paralysis of the Diaphragm

Local or Unilateral Paralysis of the Diaphragm: This may be caused by injury to the phrenic nerve, or to the spinal cord. Such paralysis is at times also the result of progressive muscular atrophy, or of the toxic action of diphtheria or of lead poisoning

Physical Signs: *Inspection* reveals dyspnea on the least exertion, reversed respiratory movements on the affected side, and diminished expansion; the observations as to diminished expansion on the affected side are confirmed by *palpation.* The complementary sinus on the affected side shows no lowering of *percussion* resonance during inspiration, and no raising during expiration On *auscultation* breath sounds are not heard below the ninth dorsal spine, even during the deepest inspiratory effort. There is usually an associated unilateral congestion of the lung

General or Bilateral Paralysis of the Diaphragm: This is a rare and extremely serious condition, which is likely to cause death if continued for any length of time. It may result from a brain lesion, a fracture of cervical vertebrae, from tumors or hemorrhage which brings about compression of the cord, or from myelitis. Not infrequently it is seen in acute poliomyelitis. It may also follow diphtheria, lead poisoning, or inflammation of the serous membrane covering the diaphragm

Symptoms: These are an inability to carry on respiration; in incomplete paralysis there are faint or incomplete respiratory movements, hiccoughing, very feeble sneezing, alteration in the voice, and the appearance of the following physical signs:

Physical Signs: *Inspection* shows cyanosis, rapid shallow breathing, reversal of the respiratory movements (the epigastrium receding during inspiration instead of bulging), and absence of the downward movement of the abdominal viscera during inspiration *Palpation* reveals decreased movement of the lower ribs; on *percussion,* the complementary spaces on both sides will be found very much diminished, while lung expansion at the bases is practically nil. *Auscultation* reveals feeble breath and voice sounds; numerous râles are usually heard at the bases of the lungs. A more accurate diagnosis of this condition can be made by radiography and fluoroscopy. X-rays will show the diaphragm arched, and little or no descent will be noted during inspiration

Pneumothorax: This signifies a collection of air in the pleural sac. Intrinsic causes are a rupture of a portion of the lung during the course of pulmonary tuberculosis, malignancy, or gangrene of the lung; extrinsic causes may be traumatism by some sharp instrument which perforates the lung, or other accident. Artificial pneumothorax is often induced as a remedial measure in tuberculosis and in gangrene and abscess of the lung. Occasionally pneumothorax of unknown origin may develop

Physical Signs: On *inspection* the patient will be somewhat cyanosed and dyspneic, bulging of the affected side will be in evidence. *Palpation* will reveal the absence of tactile fremitus, and *percussion* will yield tympany There will be absence of breath sounds on *auscultation*, except when a large communication with a bronchus exists, in which case there will be bronchial breathing. The coin test and metallic tinkling will be positive over massive pneumothorax.

Hydro- and pyopneumothorax: These are due to the presence of serum or pus in the lung in addition to air or gas

Physical Signs: Inspection will show dyspnea, cough and cyanosis, and *palpation*, the absence of tactile fremitus. *Percussion* will yield dullness over the fluid, and tympany over the air; the upper level of dullness changing with the change of the patient's posture. *Auscultation* will reveal absence of breath sounds, positive succussion splash, positive coin test and metallic tinkling. Pyopneumothorax will, in addition to these signs, also give rise to signs of sepsis, *i e.*, fever, sweats and chills.

X-ray Interpretation of Pleural Effusion, Pneumothorax and Encysted Fluid: ***Pleural Cavity: Pleural effusion*** is early recognized at the fluoroscope. In the massive effusion, extending from the base to the apex, there is almost absolute opacity on the affected side; the diaphragm is not visible;

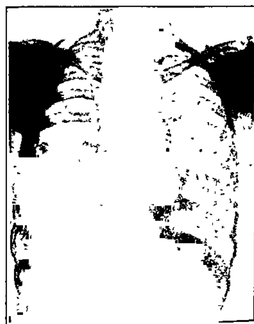


Fig. 17—Hydropneumothorax
(Courtesy Dr. Leon Solis-Cohen)

the heart is often displaced to the opposite side In a small effusion the diaphragm may be depressed and seen with difficulty The normal pulmonary transparency of the opposite side is striking In moderate effusion, the upper limit is often visualized as a line or meniscus, and by shaking the patient the splashing of the fluid can be observed. It is often possible to determine the best point for puncture of the thorax by careful screening of the patient.

Pneumothorax: The presence of an area of extreme transparency (indicative of air), replacing the normal lung shadows, is absolutely diagnostic of pneumothorax and is more or less easily determined at the fluoroscope. The air may be limited by intrapleural adhesions

and by the lung border. A partial or complete collapse of the lung may occur, according to the extent of the pneumothorax. Fluid is sometimes present as hydro- or pyopneumothorax and this will be indicated by the density of the fluid shadow beneath the transparent air

Encysted Fluid: This will show a cavity with a fluid level and a smooth, circumscribed wall. It appears to be of uniform density and air may overlie the fluid. It is quite often difficult to differentiate between an encysted empyema and a collection of pus in the intralobar areas, as both are found in contact with the pleura

Diseases of the Diaphragm

The diaphragm is normally one of the most active muscles in the body, as it constantly contracts and relaxes in its effort to carry on respiration. There are certain pathological conditions which affect the diaphragm, thereby interfering with its proper function.

Paralysis of the Diaphragm

Local or Unilateral Paralysis of the Diaphragm: This may be caused by injury to the phrenic nerve, or to the spinal cord. Such paralysis is at times also the result of progressive muscular atrophy, or of the toxic action of diphtheria or of lead poisoning.

Physical Signs: *Inspection* reveals dyspnea on the least exertion, reversed respiratory movements on the affected side, and diminished expansion; the observations as to diminished expansion on the affected side are confirmed by *palpation*. The complementary sinus on the affected side shows no lowering of *percussion* resonance during inspiration, and no raising during expiration. On *auscultation* breath sounds are not heard below the ninth dorsal spine, even during the deepest inspiratory effort. There is usually an associated unilateral congestion of the lung.

General or Bilateral Paralysis of the Diaphragm: This is a rare and *extremely serious condition, which is* likely to cause death if continued for any length of time. It may result from a brain lesion, a fracture of cervical vertebrae, from tumors or hemorrhage which brings about compression of the cord, or from myelitis. Not infrequently it is seen in acute poliomyelitis. It may also follow diphtheria, lead poisoning, or inflammation of the serous membrane covering the diaphragm.

Symptoms: These are an inability to carry on respiration; in incomplete paralysis there are faint or incomplete respiratory movements, hiccoughing, very feeble sneezing, alteration in the voice, and the appearance of the following physical signs:

Physical Signs: *Inspection* shows cyanosis, rapid shallow breathing, reversal of the respiratory movements (the epigastrium receding during inspiration instead of bulging), and absence of the downward movement of the abdominal viscera during inspiration. *Palpation* reveals decreased movement of the lower ribs; on *percussion*, the complementary spaces on both sides will be found very much diminished, while lung expansion at the bases is practically nil. *Auscultation* reveals feeble breath and voice sounds; numerous râles are usually heard at the bases of the lungs. A more accurate *diagnosis of* this condition can be made by radiography and fluoroscopy. X-rays will show the diaphragm arched, and little or no descent will be noted during inspiration

Diaphragmatitis

This is an inflammatory condition of the diaphragm which may be primary or secondary. *Primary diaphragmatitis* is a rare condition; it may result from trichinosis, and, at times, is seen in the terminal stages of scurvy. *Secondary diaphragmatitis* is frequently encountered and may result from disease of the lungs, the pleura, or the adjacent abdominal viscera

Symptoms: The chief *symptoms* are immobility of the diaphragm, a sense of constriction encircling the lower portion of the chest, dyspnea and soreness.

Physical Signs: *Inspection* shows absence of Litten's sign (phrenic wave) and diminished expansion of the lower chest. *Palpation* confirms inspection as to the limited motion of the lower chest, and *percussion* will reveal very limited complementary spaces *Auscultation* elicits very feeble breath sounds at the bases, and exaggerated breathing over the upper part of the chest

Diaphragmatic Abscess (Subphrenic Abscess)

Simple diaphragmatic abscess and gas-containing abscess are the two varieties usually found.

Simple Diaphragmatic Abscess: This may occur as a result of a ruptured displaced appendix, abscess of the liver or gallbladder, retroperitoneal abscess, abscess of the suprarenal bodies, or pyonephrosis; it also occurs in empyema and advanced pulmonary tuberculosis with basal cavity.

Symptoms: These are chills, fever and sweating, with pain in the region of the lower chest.

Physical Signs: *Inspection* will show diminished expansion on the affected side, while *palpation* confirms inspection, and reveals absence of tactile fremitus over the affected portions. *Percussion* will reveal dullness or flatness over a circumscribed area, uninfluenced by respiration or position. *Auscultation will reveal an absence of breath sounds* over the affected part

Gas-containing Abscess: The causes for this condition may be identical with those of simple abscess, the most frequent ones, however, being perforated gastric or duodenal ulcer. A subphrenic gas-containing abscess is more frequently found on the left than on the right side

Physical Signs: On *inspection* there will be diminished expansion over the lower chest and upper abdomen; *palpation* confirms inspection as to the limited motion, and reveals an absence of tactile fremitus over the affected part The apex beat may be displaced to the right in a left-sided abscess. *Percussion* reveals localized tympany, and *auscultation,* the absence of breath sounds, absence of transmitted voice sound, and negative coin test.

Diaphragmatic Pleurisy

This may be caused by a stab wound, or it may be secondary to inflammation of the pleura as a result of tuberculosis, pneumonia or empyema; *it may also* result from an inflammatory condition of any viscera lying in close proximity to the diaphragm.

Symptoms: These are slight dyspnea on exertion, pain during respiration usually referable to the epigastrium, often simulating the pain of gastric ulcer.

Physical Signs: *Inspection* shows limited diaphragmatic descent which is confirmed by *palpation. Percussion* will elicit limited diaphragmatic respiratory excursions and *auscultation* will reveal diminished breath sounds at the base,

most of the auscultatory signs, however, usually being masked by congestion at the bases of the lungs.

Hernia of the Diaphragm

This condition may be either congenital or acquired. *Congenital diaphragmatic hernia* is a condition in which the abdominal viscera and their peritoneal covering perforate a portion of the diaphragm. *Acquired diaphragmatic hernia* may be caused by a stab wound, or a sudden strain brought to bear upon the abdominal viscera which forces them upward and causes them to break through a weakened portion of the diaphragm

Symptoms: Congenital hernia seldom gives rise to any symptoms.

Acquired hernia gives rise to a sensation of sudden loss of support in the diaphragmatic region accompanied by acute pain and often by temporary collapse.

Physical Signs: The physical signs may be limited motion of the affected side on *inspection,* confirmed by *palpation,* which also reveals absence of tactile fremitus. There will be tympany on *percussion,* and on *auscultation,* absence of breath sounds, while often after taking food or drink, in the presence of a left-sided diaphragmatic hernia, splashing and gurgling sounds may be audible; these are intensified by shaking the patient (succussion).

Evisceration

This is a condition similar to diaphragmatic hernia, the two conditions often being confused It usually occurs on the left side and may be the result of an injury, such as a gunshot wound, stab wound or the result of a crushing accident or severe strain A strain, blow or crushing accident may simply tear the muscle, leaving the serous covering intact; this condition is not easily diagnosed by symptoms or physical signs, as there is no visible wound and the symptoms are often misleading.



Fig 18—X-ray plate showing diaphragmatic herniation of the stomach Note greater part of stomach is above diaphragm.

Symptoms: Those sometimes encountered in this condition are dyspnea, irritating cough, vomiting and digestive disturbances.

Physical Signs: They are those of diaphragmatic hernia

Eventration

Eventration (congenital) is rare. Some years ago, Bayne-Jones[1] collected from the literature reports of 45 cases The condition is characterized by a general expansion of one-half of the diaphragm, allowing the abdominal viscera to be displaced upward into the thoracic cavity. It is generally believed to be of

[1] Bayne-Jones: Arch. Int Med., Feb., 1916

congenital origin, and as it seldom produces symptoms is usually discovered by accident, either by roentgenography, or at the autopsy table. A case which the author saw at the United States General Hospital, No. 16, at New Haven, Connecticut, presented practically no symptoms.

Physical Signs: *Inspection* showed absence of expansion of the lower left chest, the apex beat being displaced to the right. *Palpation* confirmed inspection as to the limited motion, and revealed the absence of tactile fremitus. *Percussion* yielded tympany; when the patient was in a sitting or upright posture, the tympany extended to the fourth rib and the eighth dorsal spine (from below upward); in the prone position, after a full meal, there was dullness from the base up to the eighth rib posteriorly; tympany from the base to the fourth rib anteriorly. When the patient was fully prone, dullness could be elicited anteriorly and tympany posteriorly. The diaphragmatic movements on the left side were limited. *Auscultation* revealed that breath sounds were absent. After the patient had drunk two or three glasses of water or eaten a full meal, succussion splashes were easily elicited. The diagnosis of eventration was confirmed by Dr. Honji, who made very careful roentgenologic and fluoroscopic studies.

Displacement of the Diaphragm

The diaphragm may be displaced downward by effusion in the pleura, or upward by tumors of the abdominal viscera, enlarged glands, or dilatation of the stomach and colon

Symptoms and Physical Signs: They are of the underlying condition Causes of downward displacement of the diaphragm should be differentiated from abdominal conditions which bring about upward displacement of the diaphragm. Conditions which displace the diaphragm downward usually give signs and symptoms in the lungs, while those which bring about upward displacement will cause symptoms that are referable to the abdominal cavity.

Spasm of the Diaphragm

This condition may be an organic or functional disturbance, and may be caused by irritation of the phrenic nerve



Fig 19—Carcinoma of breast.

or, reflexly, by some disturbance of the stomach, heart, or pleurae. It may occur in hysteria and in cases of irritation of the central nervous system (apoplexy or epilepsy).

Symptoms: The most prominent symptoms of spasm of the diaphragm are hiccoughing, paroxysmal sneezing, laughing, weeping and coughing. Tonic spasms of the diaphragm sometimes occur in tetanus, strychnia poisoning and hydrophobia. The symptoms of tonic

spasm are a sense of constriction in the chest, pain along the insertion of the diaphragm, and dyspnea Physical signs are not conclusive.

Diseases of the Breasts (Mammae)

The mammae are two glandular structures situated upon the anterior chest wall between the third and sixth ribs when not pendulous. The male breast is rudimentary and in the majority of men the nipple is the only conspicuous portion of that gland. Abnormally large breasts in men, gynecomastia, may be found in the obese and in those suffering from endocrine disturbances particularly of the gonad-pituitary type.

The female breasts are fully developed glandular structures capable of lactation immediately after childbirth The size of the adult breast depends upon the corpulency of the individual, the state of lactation and personal peculiarity.

The nipple (mammilla) occupies the center of the nonpendulous breast. It contains erectile tissue and in women is perforated by lactiferous ducts.

Anomalies of the Breast—*Amazia:* The entire absence of mammary glands, may be unilateral or bilateral

Micromazia: Rudimentary breast may be unilateral or bilateral.

Polymazia (supernumerary breasts): There are reports on record of men and women who have had three, four, five and one woman had six breasts. Those in women were lactating

Polythelin (supernumerary nipples) Two or more nipples may occur upon one breast, or a rudimentary nipple may occur upon the chest wall independent of a breast The nipples may be malformed or rudimentary, preventing lactation. They may become fissured, eczematous or be infiltrated by new growths

Neuralgia of the Breast: Tender areas not accompanied by any enlargement or tissue changes may occur. The breast is usually sensitive to cold and the skin is hyperesthetic. This conditions occurs most frequently near the approach of the menstrual period

Mastitis: Inflammation of the breasts may occur at any age, but is most common during lactation because of infection through the nipple or because of trauma Women who have not borne children recently and those approaching the menopause may develop mastitis because of faulty involution. The breast becomes enlarged and develops local areas of redness which are hard and tender and may suppurate, causing a breast abscess.

Tumors of the breast may be benign or malignant

Benign Tumors: *Fibroma, lipoma, myxoma, adenoma* when located in the breast seldom give rise to pain and do not ulcerate They are not connected with the skin, so that the skin is easily moved over the tumor mass Benign tumors usually occur in young adults and do not give rise to metastasis

Malignant Tumors: These are sarcoma and carcinoma.

Sarcoma: The type of sarcoma depends upon its embryonic cell formation, *i. e.,* round cell, spindle cell, myeloid, lymphoid, etc These tumors often attain a large size, they have a tendency to ulceration and give rise to metastasis

Carcinoma: This is the commonest and the most fatal of the breast tumors It usually affects women near the menopause, though it may occur at any age The tumor mass is, as a rule, located near the nipple, causing retraction. It

is adherent to the skin, causing puckering, the skin is not movable over the mass and is tender to touch. The lymphatic gland becomes enlarged and metastasis occurs early. Massive destruction of tissue occurs in advanced cases.



Fig 20—Carcinoma of breast (Philadelphia General Hospital.)

Cysts: Cysts of the breasts may occur at any age; they may be single or multiple and may contain various substances.

Galactocele is a cyst containing milk This may be found during lactation; the breast becomes hard and a large rounded noninflammatory fluctuating mass makes its appearance. Involution cysts are usually multiple and occur during the involution period of the breasts. The masses are hard, painless and are noninflammatory.

Hydatid cysts are rare. They usually attain a large size, are noninflammatory and, as a rule, do not cause pain

The diagnoses of cysts and other tumors are more accurately made microscopically; therefore the presence of a suspicious tumor upon the breast should be treated surgically. It is far better to remove a hundred "innocent" tumors from the breast than to leave one that may be malignant.

Mazoplasia (adenosis): This is a hyperplasia of the breast confined chiefly to the alveolar system. It is palpable as small hardened masses, and may be associated with bleeding from the nipple. This is often found in women at the menopause or in those suffering from ovarian disease, who have an overproduction of anterior pituitary sex hormone with a deficiency of ovarian hormone.

Cystic Hyperplasia: This is characterized by the formation of round freely movable masses in the center of the breast. These originate in the duct system and may be single or multiple. This condition is said to be due to the uninterrupted production of large amounts of estrin by persistent ovarian follicle cysts

Paget's Disease of the Nipple: This is a crusting ulceration or erosion with retraction of the nipple. Dislodging of the crusts exposes a raw surface which may bleed. Occasionally there is a serosanguineous discharge from the nipple This condition is considered to be malignant

Diseases of the Mediastinum[1]

The mediastinum, because of its position and its contents, is vulnerable to various affections such as acute simple, acute suppurative, chronic indurative and

[1] For Anatomy of the Mediastinum, see p 383

chronic suppurative mediastinitis (abscess), adenitis, neoplasms, cysts, aneurysm, emphysema and hemorrhage.

Acute Simple Mediastinitis: This is an acute inflammation of the mediastinal connective tissue without suppuration It may be caused by injury or disease of the ribs, the sternum, the inner aspects of the clavicles, the intercostal muscles, or the thoracic vertebrae It may also result from wounds of the esophagus or trachea Occasionally it may be secondary to inflammation of the pleura, lungs, pericardium or peritoneum.

The clinical manifestations are vague so that the diagnosis is often overlooked There may be a sense of heaviness or crowding in the anterior chest or some tenderness on pressure over the sternum, occasionally there may be heard fine crepitations over the sternum during deep respiration or synchronously with the heart beat There may also be a short hacking nonproductive cough brought out by deep breathing or by talking, and there is usually a slight rise in temperature. In the absence of complications recovery usually takes place within one week.

Acute Suppurative Mediastinitis or Abscess: This may follow the acute simple type or start as an acute infective suppurative process secondary to infection of the chest wall, the spine or the mediastinal contents It may also result from blood stream infections, erysipelas, actinomycosis, infections about the face, mouth or neck, and from empyema and pyopericardium Occasionally it may be a complication in influenza, typhoid fever. pneumonia, pneumothorax, tuberculosis, syphilis, lymphogranuloma and other severe infections.

Clinical Manifestations: If the suppurative process in the mediastinum develops during the febrile stage of an acute infection, it may remain undiagnosed. Its presence may however be suspected by the occurrence of chills, an increase in temperature that may show a definite septic curve, sweats, and severe retrosternal pain with a sense of suffocation. When the abscess is circumscribed and large it may cause signs of tumor, that is, partial bronchial stenosis, difficulty in swallowing, venous distention and other signs of the mediastinal syndrome.

Fluoroscopic examination in the anteroposterior and lateral positions may reveal the abscess, and x-ray plates taken in these postures may show the encroaching shadow. Abscess of the mediastinum is fatal in most instances Recovery may occur if the abscess points at the surface and is aspirated, or when it ruptures into a bronchus and does not cause suffocation

Chronic Indurative Mediastinitis: This may be a sequel to the acute types It is usually evidenced by mediastinal fibrosis, pericardial adhesions often with adhesive bands compressing the great vessels and is occasionally associated with caseation of the mediastinal lymph glands.

Mediastinopericarditis is characterized by great cardiac hypertrophy with dilatation causing cyanosis, dyspnea, cough, portal and renal congestion and occasionally perisplenitis, perihepatitis and ascites known as pericarditic pseudocirrhosis or Pick's disease

Clinical Manifestations: These are referable mainly to the associated lesions of the heart and pericardium with tenderness over the sternum. The findings are those of the inferior mediastinal syndrome.

Chronic Suppurative Mediastinitis (Chronic Abscess): This is generally due to caseation of tuberculous peri-

bronchial or mediastinal glands, or to tuberculosis of a spinal vertebra. It may also be caused by a foreign body such as a needle or bullet lodged in the tissues of the mediastinum The clinical manifestations depend upon the size of the abscess and the absorbability of the pus A large abscess will cause pressure symptoms. Slow absorption of the pus will cause mild toxic symptoms characteristic of a cold abscess.

Mediastinal Adenitis: Normally the mediastinal lymph nodes are situated in the anterior and posterior mediastinum and principally around the bifurcation of the trachea and along the bronchi. Enlargement and inflammation of any group or of all of them may result from infection of the structure that is drained by them Mediastinal adenitis may also occur in tracheobronchitis, influenza, the pneumonias (virus, atypical or pneumococcic), pertussis, measles, pneumonoconiosis, malignancy of the lungs or bronchi, tuberculosis, syphilis, lymphatic leukemia, Hodgkin's disease, acute mononucleosis, lymphogranuloma, and other conditions that have a predilection for lymphoid tissue

Clinical Manifestations: When the glands are small they may escape detection. When of moderate size, D'Espins sign and a venous hum may occasionally be detected over the manubrium (Eustace Smith's sign). When large, the "mediastinal syndrome" may be elicited The diagnosis of tuberculous adenitis is particularly important in children. The child usually runs a prolonged subfebrile temperature, 99° to 100°, over a period of months, has a lymphocytosis, a slight dry hacking cough and appears pale and ill nourished The appetite is poor; there is generally irritability, and often night sweats. Dilated veins are seen over the upper part of the chest, anteriorly and posteriorly, and there may be inequality of the pupils.

Tumors and New Growths of the Mediastinum: These may be benign or malignant. The benign tumors found in this location are retrosternal goiter, persistent thymus, lipoma, fibroma, chondroma, osteochondroma, myoma, intrathoracic adenoma and cysts, such as echinococcic and dermoid, and aneurysm These tumors, when small, may be overlooked except by x-ray. When they are large enough to exert pressure they may be detected by the mediastinal syndrome.

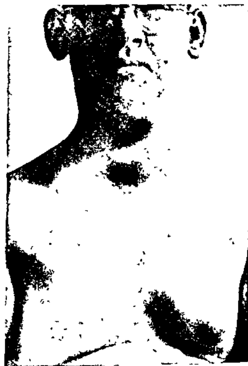


Fig 21—Mediastinal tumor showing edema and cyanosis of face and neck (*Jefferson Hospital, courtesy of Dr. H. A Reimann*)

Malignant Neoplasms: These may be primary, arising from the mediastinal tissue, or they may be secondary due to extension or metastasis from adjacent or distant organs. The neoplasms encoun-

tered in this region are carcinoma, various types of sarcoma and thymoma

Clinical Manifestations In addition to the usual signs encountered in malignancy, there are mediastinal pressure symptoms, the so-called mediastinal syndrome.

The Mediastinal Syndrome: This syndrome consists of a group of symptoms and physical signs caused by compression of the various structures within the mediastinum It is often divided into the superior and the inferior mediastinal syndrome according to the area of maximum compression.



Fig 22—Same as Fig 21 showing collar of Stokes

The Superior Mediastinal Syndrome: This is manifested by the following symptoms and physical signs brought about by pressure upon the venae cavae, the vagus, the sympathetics, the recurrent laryngeal nerves, the esophagus, and the trachea.

Symptoms: These are· (1) Pain in the sternal region and base of the neck may be sharp, dull or oppressive; it is aggravated by deep breathing, talking or walking (2) Hoarseness is of a peculiar harshness. (3) Cough is often persistent, has a brassy quality and may be dry, or there may be various amounts of sputum, the kind and quality depending upon the accompanying bronchial and pulmonary inflammation (4) Dyspnea, associated with a wheeze, is due to tracheal or bronchial compression. (5) Dysphagia is due to pressure upon the esophagus (6) Paralysis of one side of the diaphragm is caused by compression of the phrenic nerve.

Physical Signs: *Inspection* (1) Posture The patient usually prefers to lean forward and when he sits erect the head is held in hyperextension (2) Cyanosis of the head, neck and upper chest The cyanosis terminates abruptly revealing a sharp line or demarcation (collar of Stokes) (3) Marked venous distention of the head, neck, upper thorax and upper extremities This may be accompanied by edema *Palpation* This will elicit tenderness over the upper sternum, clavicles and ribs *Percussion* Dullness is elicited over the upper sternum and at times in the upper part of the intrascapular region. *Auscultation* Various crunching sounds, sibilant and sonorous râles may be audible when there is partial pulmonary compression In complete compression of a bronchus breath sounds are absent

Lower Mediastinal Syndrome: This is caused by pressure upon the esophagus,

bronchial or mediastinal glands, or to tuberculosis of a spinal vertebra. It may also be caused by a foreign body such as a needle or bullet lodged in the tissues of the mediastinum The clinical manifestations depend upon the size of the abscess and the absorbability of the pus A large abscess will cause pressure symptoms. Slow absorption of the pus will cause mild toxic symptoms characteristic of a cold abscess.

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Malignant Neoplasms: These may be primary, arising from the mediastinal tissue, or they may be secondary due to extension or metastasis from adjacent or distant organs The neoplasms encoun-

SECTION 7

The Cardiovascular System

inferior vena cava, hepatic veins and the heart. The symptoms and signs are dysphagia, enlargement of the liver, ascites, distended veins over the abdomen and lower extremities, edema of the legs and a higher blood-pressure reading in the lower extremities than in the upper extremities.

Aneurysm: Aneurysm of the aortic arch may cause the kind of pressure symptoms found in solid tumors or large mediastinal glands, particularly so when thrill, bruit and tracheal tug are not demonstrable This is particularly true of saccular aneurysm Fluoroscopic examination may show pulsation and the *x-ray will usually reveal a comparatively* small heart in aneurysm and a much larger heart in most of the other mediastinal tumors. The history and other findings may also help in the differentiation between aneurysm and solid tumor (See pp. 531 and 535).

Mediastinal Emphysema: Mediastinal emphysema may be caused by artificially induced pneumothorax or by spontaneous pneumothorax, by a penetrating wound, by erosion of the esophagus, the trachea or a main stem bronchus, and by inflammatory lesions in the neck. *The clinical manifestations* are sudden retrosternal pressure and dyspnea followed by subcutaneous emphysema in the neck and chest and tympany replacing sternal and heart dullness Breath sounds and heart sounds may be inaudible over the anterior chest wall.



Fig 23—X-ray plate of Fig 21. Showing mediastinal tumor due to Hodgkin's disease.

Mediastinal Hemorrhage: *This may* result from a fractured sternum, penetrating wound, ruptured aneurysm, or other blood vessel in that region. Small hemorrhages may pass undetected. Large hemorrhage if spontaneous will cause sudden oppression in the anterior chest, small rapid pulse, *dyspnea, and signs of* internal hemorrhage associated with the mediastinal syndrome.

CHAPTER XV

Anatomy and Physical Examination of the Heart

The cardiovascular system is concerned with the circulation of the blood throughout the body. Since the functional ability of all the organs and tissues is largely dependent upon an adequate circulation, it is important, when examining the cardiovascular system, to study not only the heart and blood vessels but also the individual as a whole and the various organs that are directly or indirectly concerned with the circulation

When eliciting a history, cognizance is to be taken of such symptoms as cough, dyspnea, intolerance to exercise, digestive disturbances, headache, dizziness, chest pains, pain in the extremities, etc The previous medical history should be inquired into since such diseases as rheumatic fever, chorea, tonsillitis, diphtheria, syphilis, as well as other infections and hyperthyroidism often have a crippling effect upon the cardiovascular system It is important to learn of the habits and the occupation of the individual since these often leave their imprint upon the heart and blood vessels. Nor may the question of heredity be overlooked, because in certain families or clans cardiovascular disease is fairly prevalent. In eliciting the physical signs of the patient attention should be directed to the color of the skin and mucous membranes, the presence of distended veins or of unusually throbbing arteries, and the presence of dyspnea. The lungs should be carefully examined for signs of passive congestion, edema and pleural effusion. The abdomen is to be examined for the presence of ascites and enlargement of the liver and spleen The extremities are to be examined for signs of cyanosis, edema, and the condition of their vessels. When a complete history has been elicited and a careful general physical examination has been done, then special attention is to be directed to the physical examination of the heart.

Inspection is the first procedure by which the position and extent of the apex beat is to be noted *Palpating* the precordium for the location and extent of the apex beat and other pulsations or thrills is the next step *Mensuration* is done to determine accurately the distance of the apical impulse from the midsternal line. It is a measure of refinement in diagnosis and should be carried out. *Percussion* should be practiced so as to determine fairly accurately the size of the heart and the extent of cardiac dullness. *Auscultation* of the apex beat and at the cardiac valve areas should be practiced diligently so as to determine the quality, rate and rhythm of the heart sounds and the presence of adventitious sounds The study of the pulse and the arterial and venous systems must be included in every cardiovascular examination. Under certain circumstances an electrocardiographic tracing and an x-ray study of the heart may become necessary These studies should not displace a thorough physical examination because no instrument has as yet been invented which is as capable of evaluating the patient as a whole and his relation to his cardiovascular system as can

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Anatomy and Physical Examination of the Heart

The cardiovascular system is concerned with the circulation of the blood throughout the body. Since the functional ability of all the organs and tissues is largely dependent upon an adequate circulation, it is important, when examining the cardiovascular system, to study not only the heart and blood vessels but also the individual as a whole and the various organs that are directly or indirectly concerned with the circulation

When eliciting a history, cognizance is to be taken of such symptoms as cough, dyspnea, intolerance to exercise, digestive disturbances, headache, dizziness, chest pains, pain in the extremities, etc. The previous medical history should be inquired into since such diseases as rheumatic fever, chorea, tonsillitis, diphtheria, syphilis, as well as other infections and hyperthyroidism often have a crippling effect upon the cardiovascular system It is important to learn of the habits and the occupation of the individual since these often leave their imprint upon the heart and blood vessels Nor may the question of heredity be overlooked, because in certain families or clans cardiovascular disease is fairly prevalent In eliciting the physical signs of the patient attention should be directed to the color of the skin and mucous membranes, the presence of distended veins or of unusually throbbing arteries, and the presence of dyspnea The lungs should be carefully examined for signs of passive congestion, edema and pleural effusion The abdomen is to be examined for the presence of ascites and enlargement of the liver and spleen. The extremities are to be examined for signs of cyanosis, edema, and the condition of their vessels. When a complete history has been elicited and a careful general physical examination has been done, then special attention is to be directed to the physical examination of the heart.

Inspection is the first procedure by which the position and extent of the apex beat is to be noted *Palpating* the precordium for the location and extent of the apex beat and other pulsations or thrills is the next step. *Mensuration* is done to determine accurately the distance of the apical impulse from the midsternal line. It is a measure of refinement in diagnosis and should be carried out *Percussion* should be practiced so as to determine fairly accurately the size of the heart and the extent of cardiac dullness *Auscultation* of the apex beat and at the cardiac valve areas should be practiced diligently so as to determine the quality, rate and rhythm of the heart sounds and the presence of adventitious sounds The study of the pulse and the arterial and venous systems must be included in every cardiovascular examination Under certain circumstances an electrocardiographic tracing and an x-ray study of the heart may become necessary. These studies should not displace a thorough physical examination because no instrument has as yet been invented which is as capable of evaluating the patient as a whole and his relation to his cardiovascular system as can

be done by a thorough physical examination It must also be borne in mind that various laboratory tests are often required as an aid in determining the *etiology and the prognosis* of the cardiovascular patient.

Anatomy

The Mediastinum

The mediastinum is a space formed in the midline of the thoracic cavity by *the approximation of the two deflected* pleurae; it divides the chest into two pulmonary cavities. The two pleurae are not, however, in contact with each other at the midline, but have a space between *them, which contains all the chest viscera*, except the lungs. The mediastinum is divided into *anterior, superior* and *middle regions.*

1. *The anterior mediastinum* which *lies in front of the heart, and in contact* with the sternum, between the second and sixth ribs inclusive, has an upper part which is narrow and shallow (above the fourth rib) and a lower part corre*sponding to the quadrilateral free space* Its contents are unimportant.

2. *The superior mediastinum* is the section above the heart containing the trachea, the esophagus, the thoracic duct, *the transverse portion of the aortic arch,* the innominate artery, the left carotid, subclavian and innominate veins, the upper part of the superior vena cava, the two pneumogastrics, the left recur*rent laryngeal, the phrenic and cardiac* nerves, the thymus gland or its remains, and some bronchial and lymphatic glands.

3. *The middle mediastinum* contains the pericardium and its contents, *viz.,* the heart, the ascending portion of the arch of the aorta, the pulmonary artery and vein and *the lower half of the superior vena cava*; also the phrenic nerve and vessels, the termination of the azygos vein, the bifurcation of the trachea, and some bronchial lymphatic glands.

The Precordium

The precordium is a rectangular, arbitrarily-defined space overlying the heart, its great vessels and the pericardium. It is bounded above by the second rib; below by the sixth rib; its right boundary is the right parasternal line, and *its left boundary the left* midclavicular line.

The Pericardium

The pericardium is a cone-shaped, *fibroserous sac which occupies the middle* mediastinum, and contains the heart and the roots of the great blood vessels It is attached by its broad base to the diaphragm, while its apex extends upward by diverticulae upon the walls of the great vessels as far as their first subdivision. It is also attached in front *to the sternum; laterally, to the mediastinal* pleura, and posteriorly, to the esophagus, trachea and the main bronchi The phrenic nerve passes over its lateral surface.

The Heart

This double-cycle pump of the body's vital fluid is irregularly pyramidal in shape, and *is situated in the mediastinum*. Its weight varies with the stature of the individual, general musculature and sex. In the male, it ranges from 250 grams to 350 grams (7.5 to 10.5 oz.); in *the female, from 200 to 300 grams* (6 to 9 oz.), and in children, proportion-

ately to their age and physical development. The heart measures from 11 to 13 cm. (4⅓ to 5½ inches) in length, from 7½ to 9½ cm. (3 to 3¾ inches) in breadth and 5½ to 6½ cm. (2⅛ to 2½ inches) in thickness. Its size may roughly be compared to that of its owner's fist. It is freely movable within the pericardial sac, its only attachment being the great vessels which originate from its base. It rests upon the central tendon of the diaphragm

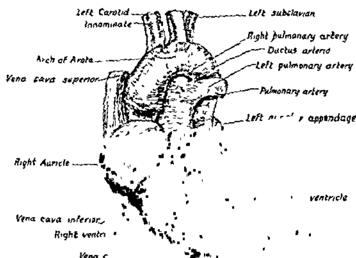


Fig 1—Anterior view of the right chambers of the heart with the great vessels.

The *base* of the heart is directed upward, backward and toward the right, and is on a level with the second intercostal space.

The *apex* of the heart points downward, forward and to the left, on a level with the fifth intercostal space beyond the parasternal line or about 8 to 9 cm. (3¼ to 3½ inches) to the left of the midsternal line. The long axis of the heart is inclined at an angle of 60° to the body.

The right auricle and ventricle, a small portion of the left ventricle (the left edge) and the apex of the heart are anteriorly situated. Anteriorly the heart is almost entirely covered by the lungs and only a small quadrilateral portion of the right ventricle is exposed to the anterior chest wall. This exposure is caused by the recession of the anterior border of the left lung at the fourth rib and interspace.

The Heart Chambers: The heart contains four chambers or cavities, two chambers to each side of the heart, an upper (auricle or atrium) and a lower (ventricle). These are designated respectively as right auricle and right ventricle and left auricle and left ventricle. The two auricles lie uppermost and constitute the base of the heart, these chambers are smaller and their muscular walls are thinner than their respective ventricles. The left ventricle is larger and its wall thicker than the right ventricle There is no intercommunication in the normal heart (after birth) between the auricles and none between the ventricles. Each auricle communicates

with its respective ventricle through an orifice which is guarded by a valve, known as the auriculoventricular valve. The *mitral* or *bicuspid valve* separates the *left* auricle from the left ventricle.

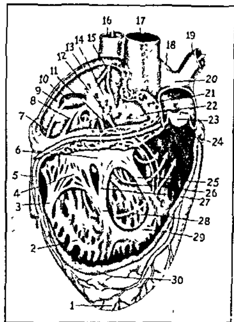


Fig 2—Right auricle and ventricle Both chambers laid open, the anterior wall of each having been removed The arrows indicate the course of the blood 1, Apex

opening of superior vena cava, 16, superior *vena cava*, 17, *aorta*, 18, *right* branch *of* pulmonary artery, 19, left branch of pulmonary artery; 20, pulmonary artery; 21, pectinate muscles; 22, auricular appendix; 23, posterior flap of pulmonary vagus;

The *tricuspid valve* separates the *right* auricle from the right ventricle.

Each auricle has also a second orifice (valveless) through which the left auricle receives blood from the pulmonary veins, and the right auricle receives blood from the superior and inferior venae cavae for transference to their respective ventricles through the auriculoventricular orifices The ventricles in turn propel the blood thus received, through orifices which are guarded by valves (the semilunar valves), into the aorta by the left ventricle and into the pulmonary artery by the right ventricle

The *aortic valve* guards the orifice between the left ventricle and the aorta

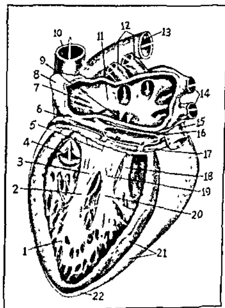


Fig. 3—The left auricle and ventricle

cardiac vein, 7, pectinate muscles, 8, auric-

artery; 14, left pulmonary veins, cava inferior, 16, coronary sinus, 17, transverse branch of the right coronary artery; 18, papillary muscles of the posterior flap, 19, *chordae tendinae*; 20, papillary muscles; 21, muscular wall; 22, apex

The *pulmonic valve* guards the orifice between the right ventricle and the pulmonary artery

Anatomical Position of the Heart Valves: The anatomical position of the

four heart valves is situated in a space bounded by the third and fifth ribs and the sternum. This differs greatly from their *clinical* position

The *pulmonary valve*, guarding the pulmonic opening, lies uppermost, it is.

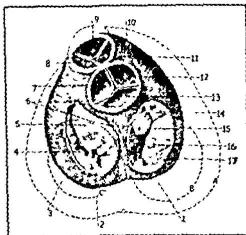


Fig 4—The valves of the heart. View from above, showing their relative size and position during systole The dotted lines indicate the respective sizes during the rest period (Spaltehoiz) 1, Ventriculus dexter, 2, annulus fibrosus sinister; 3, ventricular sinister; 4, cuspis posterior valvulae bicuspidalis (mitralis); 5, cuspis anterior valvulae bicuspidalis (mitralis); 6, trigona fibrosa, 7, valvula semilunaris sinistra aortae; 8, valvula semilunaris sinistra a. pulmonalis; 9, valvula semilunaris anterior a. pulmonalis, 10, conus arteriosus; 11, valvula semilunaris dextra a. pulmonalis, 12, valvula semilunaris dextra aortae, 13, valvula semilunaris posterior aortae, 14, cuspis anterior; 15, cuspis medialis, 16, cuspis posterior (14, 15, and 16 make up the valvulae tricuspidalis); 17, annulus fibrosus dexter

situated directly beyond the upper part of the left third costosternal articulation

The *aortic valve*, guarding the aortic opening, is more centrally located than the pulmonary valve It is on a level with the third intercostal space behind the sternum, somewhat to the left of the midsternal line.

The *mitral valve* (between the left auricle and ventricle) lies on a level with the fourth rib and interspace behind the sternum, a little to the left of the median line.

The *tricuspid valve* (between the right auricle and ventricle) is in the median line behind the sternum It is on a level with the fourth interspace and the fifth rib

Clinical Positions of the Valves: That is, the points at which the sounds are best heard, are:

Pulmonary: Second interspace to the left of the sternum

Aortic: Second interspace to the right of the sternum.

Mitral: At the apex beat (fifth interspace, 2.5 cm. or one inch to the right of the left midclavicular line).

Tricuspid: At the right border or center of the lower end of the sternum.

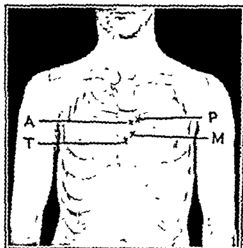


Fig 5—Anatomical position of heart valves.

The above mentioned areas are clinically chosen because the sounds produced by the various valves in closing can be heard with the greatest intensity at those points

Topographic Outline of the Heart: The exact position of the heart varies

in different individuals, and often, in the same individual at different times. This is particularly true of its lower border. The heart is held *in position chiefly* because of its suspension from the great vessels, this being the only fixed point. It rests upon the central tendon of the

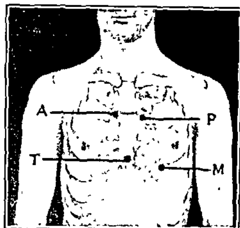


Fig 6—Points indicating clinical position of the heart valves

diaphragm, which acts only as a support having no attachment to the heart Therefore, when the diaphragm is pushed downward as in forcible inspiration, the heart descends lower, and *per contra*, during deep expiration, the diaphragm raises it considerably. The type of chest should be borne in mind when the position of the heart is considered, because differences in the length and width of the chest will alter the position of the heart in its relation to the chest wall

Change in the position of the body alters the position of the lower portion of the heart, as it will gravitate toward the dependent portion of the body. The upper boundary of the heart is more nearly constant. In children the heart hangs higher than in adults, probably because of the greater arching of the diaphragm and the proportionately shorter vessels. In the aged, the heart extends about one interspace lower than in the young adult, no doubt because of the laxity of the diaphragm and the stretching of its upper attachment.

The average position of the heart may be described as follows:

The *upper border* corresponds to a line drawn through the upper edge of the third costal cartilage, extending 1.25 cm. or ½ inch to the right of the right sternochondral articulation, and 2.5 cm. or one inch to the left of the left sternochondral articulation. This line forms *the clinical base of the heart*, passing through the tops of the auricles; it acts as the dividing line between the auricle

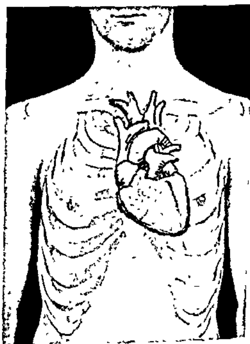
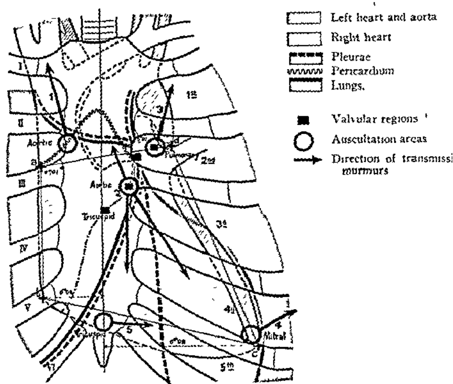


Fig 7—Position of the heart, aorta and the great vessels in relation to the anterior chest walls and ribs.

and the great vessels. The highest point of the heart is the left auricle, reaching the second intercostal space near its sternal articulation.

The *lower border corresponds* to a slightly curved line drawn obliquely



PROJECTION OF THE MORE IMPORTANT CARDIAC LANDMARKS OF THE CHEST WALL

The accompanying Figure is intended to show the relationship existing between the outer wall of the thorax and the thoracic viscera, i e, the relations of surface to depth in this part of the body

The osseous sternocostal and cartilaginous framework is shown in white on a colored background, and comprises I, II, III, IV, V, and VI, referring respectively to the 1st, 2nd, 3rd, 4th, 5th, and 6th ribs, and 1st, 2nd, 3rd, 4th, and 5th, referring respectively to the 1st, 2nd, 3rd, 4th, and 5th costal interspaces

The pleural *culs-de-sac* are outlined by the broken red lines

The attenuated anterior borders of the lungs are outlined by the solid red lines In a general way the red color refers to the lungs in a state of deep inspiration

The heart and great vessels are shaded gray

Recollection of these anatomical facts is indispensable for accurate interpretation of the results of many methods of cardiopulmonary examination, particularly percussion, auscultation, and fluoroscopy. They enable the examiner to understand, without further investigation, the mode of production of many extracardiac murmurs and their subordination to the respiratory movements, the changes in the fluoroscopic shadows and heart dullness in left-sided cardiac hypertrophy (of the ox-heart type in interstitial nephritis) and in dilatation of the right auricle in the presence of marked cardiac weakness, the location and radiation of many precordial pains, etc

The projections on the chest wall of the valvular regions, of the points for auscultation of the mitral, aortic, tricuspid, and pulmonary valves, and of the mean direction of transmission of the various murmurs should be carefully noted

across the chest with its convexity downward from the apex (fifth interspace inside the midclavicular line), across the base of the ensiform cartilage to a point 2 5 cm. or one inch to the right of the right sternal line in the fifth interspace. This border is formed by the right ventricle and apex of the left ventricle (anatomical base, not clinical)

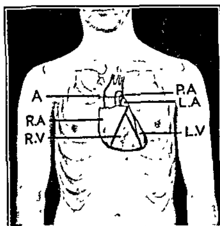


Fig. 8—The anterior aspect of the normal heart and great vessels, showing their relation to the anatomical landmarks (ribs), sternum (clavicles), of the front of the thorax

The *right border* is indicated by a slightly curved line (to the right) uniting the upper right with the lower right points (third rib 1.25 cm or ½ inch to the right of the sternal articulation to a point 2 5 cm or one inch to the right of the right sternal line in the fifth interspace) This border is formed by the right auricle

The *left border* coincides with a slightly convex line (to the left) joining the cardiac apex with the upper border 2.5 cm or one inch to the left of the third sternochondral articulation. This border is formed by the left ventricle

The *auriculoventricular septum* corresponds to a line drawn across the sternum from the third left to the seventh right sternochondral articulation.

The *interventricular septum* is indicated by a line drawn from the third left sternal articulation to a point inside the apical area

The Blood Supply of the Heart: Though the entire quantity of the body's blood passes through the heart several times an hour, it does not utilize the blood for its own nutrition unless it is brought to it by the cardiac blood vessels, among which, the *coronary arteries* are the most important.

The left side of the heart is supplied largely by the left coronary artery which arises from the left aortic sinus, dividing into a circumflex branch which supplies the left ventricle and auricle; and a left descending branch which runs along the anterior longitudinal sinus towards the apex of the heart, supplying the interventricular septum, the left ventricle and to a slight extent the right ventricle.

The right side of the heart is supplied largely by the right coronary artery which arises from the right aortic sinus It lies between the right auricle and conus arteriosus along the posterior longitudinal sulcus, and as the posterior descending ramus it almost reaches the cardiac apex. Branches of the right coronary artery supply the right auricle, the right ventricle, and to some extent also the left ventricle. The coronary arteries anastomose freely by means of minute branches, thereby establishing a collateral circulation if one of the branches should become occluded. The veins and thesbian vessels may also assist in the cardiac nutrition (Bellet).

The *veins* of the heart accompany the arteries and empty directly into the right auricle.

The *lymph* vessels of the heart are numerous. They originate from the lymph spaces in the clefts between the muscle fibers, run parallel to the blood vessels and terminate in the thoracic and right lymphatic ducts

Nerve Supply of the Heart: The heart possesses an extrinsic and intrinsic innervation. The extrinsic innervation consists of (*a*) the sympathetic cardiac nerves, (*b*) the cardiac branches of the vagi, and (*c*) the cardiac plexus.

(*a*) The *sympathetic cardiac nerves* consist of the *superior, middle,* and *inferior cardiac nerves,* arising from the superior, middle and inferior cervical ganglia respectively, and several branches which arise from the *sympathetic trunk* below the inferior cervical ganglion. The function of the sympathetic cardiac nerves is *"cardiac acceleration."*

(*b*) The *cardiac branches of the vagi* consist of three branches on either side, namely, *the superior cervical; the inferior cervical,* which is the largest cardiac branch of vagus origin; and the *thoracic cardiac branch* which arises from the vagus trunk within the thorax. The function of the vagus cardiac nerves is *"cardiac retardation."*

(*c*) The *cardiac plexus* is situated at the base of the heart and consists of a superficial and a deep part It is composed largely of the various cardiac nerves of vagus origin with but a meager sympathetic supply. Its various ramifications are found at the base of the heart, the pericardium, the aortic arch, the coronary vessels and the larger veins The cardiac plexus is supposed to assist in the *regulatory control* of heart rhythm.

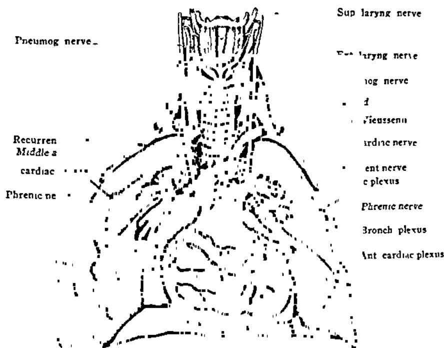


Fig. 9—The nerves of the heart

The intrinsic innervation of the heart is through a widely distributed chain of ganglia containing neurons largely of parasympathetic origin (Kuntz). The functions of the intrinsic cardiac nervous

system are not fully known. It seems to have a regulatory control which is exercised through the visceral components of the cerebrospinal nerves involved in the innervation of the heart, as cardiac rhythm is not entirely dependent on the central nervous system. (For origin of the cardiac impulse, SEE: p. 424).

The supracardiac vascular area comprises a rectangular space extending from the cardiac base to the clavicles, and bound on either side approximately by the parasternal lines. Within this area are found the aortic arch, the superior vena cava, the innominate artery and veins.

The Great Vessels of the Heart

The Aorta: The aorta arises from the base of the left ventricle, ascends a short distance, then arches backward and to the left to descend on the left side of the vertebral column. The ascending aorta lies behind the sternum. It originates near the third left chondrosternal articulation and ends at the second right costal cartilage The aortic arch commences at the second right costal cartilage running obliquely upwards and backwards towards the fourth thoracic vertebra, where it becomes the descending thoracic aorta. The highest point of the aortic arch is at the center of the sternum, usually about one inch (2.5 cm) below the suprasternal notch.

The innominate artery arises from the right upper part of the aortic arch and runs obliquely upward to the right sternoclavicular junction where it divides into the right subclavian and common carotid arteries.

The left subclavian and common carotid arteries arise from the aortic arch between its middle and posterior extremities (left), the subclavian runs almost vertically upwards into the neck and the common carotid runs obliquely upwards into the neck.

The Innominate Veins: The right lies under the inner extremity of the right clavicle, and the left lies beneath the upper portion of the manubrium.

The Superior Vena Cava: This begins at the junction of the innominate veins at the right sternoclavicular articulation and runs parallel to the sternum, lying beneath and somewhat external to its right border, and ends at the third chondrosternal articulation (its entrance into the right auricle).

The Pulmonary Artery: This runs along the left sternal border beneath the second intercostal space and the second costal cartilage.

Physical Examination

Inspection

Having by general examination previously ascertained the posture of the patient, his color, the presence or absence of cyanosis, edema, dyspnea, distended veins, abnormally pulsating vessels, etc., the examiner may now confine his attention to local inspection of the heart area.

Technic: The anterior surface of the chest is bared of all clothing and the patient is placed in a position where a good light will fall upon the part to be examined. During the examination the patient may be standing, sitting or lying flat upon his back, depending upon the severity of his condition Often all three positions are utilized in the examination of the same patient The examiner should always handle the patient gently so as to gain his confidence and avoid any ex-

citement. Inspection of the heart is practically confined to the precordial area, and to visible pulsation in the superficial vessels.

Purpose: The *object* of cardiac inspection is to observe: (*A*) The general contour and appearance of the precordium, and particularly the presence of abnormal bulgings or depressions; (*B*) abnormal pulsation in the precordial area and in the neck and extremities, and (*C*) the location, force and extent of the apex beat.

A. Contour and Appearance of the Precordium

1. *Abnormal* precordial *prominence* or bulging may be caused by the following conditions:

(*a*) Swelling of the cellular tissue or by fatty tumor.

(*b*) Undue prominence of the ribs, caused either by rickets or by a badly united fracture

(*c*) Deformity of the chest due to spinal curvature.

(*d*) Hypertrophy of the heart from any cause, particularly in very young subjects.

(*e*) Pericardial effusion and huge left-sided pleural effusion in thin-chested individuals.

(*f*) Aneurysm.

(*g*) Mediastinal tumors (usually seen above the fourth rib).

(*h*) Tumor of the ribs, sternum or chondral cartilages.

2. *Abnormal* precordial *depressions* may be caused by:

(*a*) Scoliosis and rachitic or occupational deformities.

(*b*) Unilateral chronic pleural adhesions; adhesions between the pleurae are usually very strong and their contraction is gradual. Such contraction, particularly if associated with partial pulmonary collapse, will draw the ribs inward, thus producing the deformity; pulmonary cavity in the proximity of the precordium will have a like effect.

(*c*) Adherent pericardium; in this instance the chest wall is prevented from expanding because of adhesions between the pericardium and the parietal pleura; disuse of the intercostal muscles may result in slight atrophy, thus causing the general contour of the chest to be lost, and will produce a depression.

B. Precordial Pulsations (Other Than the Apex Beat)

1. *Pulsations at the base of the heart* may be caused by:

(*a*) Hypertrophy of one or both auricles.

(*b*) Retraction of the lung or pulmonary cavity in that part of the lung which covers the auricles.

(*c*) Aneurysm of the arch of the aorta

(*d*) Mediastinal tumor in close proximity to the aorta.

(*e*) Diffuse pulsation over the entire heart area, often seen in individuals with very thin and emaciated chest walls.

2 *Epigastric pulsation* may be caused by:

(*a*) Rapid heart action from any cause.

(*b*) Dilated right ventricle resting upon the diaphragm. The exaggerated impulse of the heart is transmitted to the diaphragm because of its close proximity. The diaphragm in turn transmits this impulse to a portion of the anterior abdominal wall, the epigastrium.

(*c*) Pulsating liver (*i. e*, tricuspid regurgitation).

(*d*) Pulsating aorta, often seen in neurotic individuals with a thin belly wall

(*e*) Aneurysm of the abdominal aorta.

(*f*) Pulsating empyema.

(*g*) Tumors on the left lobe of the liver; transmitted pulsations from the aorta through the pyloric end of the stomach, the pancreas or enlarged lymph glands resting upon the aorta.

(*h*) A greatly displaced heart.



Fig 10 Broadbent's sign Adherent pericarditis showing systolic retraction

3. *Pulsations in the right axillary region* may be caused by:

(*a*) Transposition of the heart to the right side.

(*b*) Pulsating empyema.

(*c*) Aneurysm of the arch of the aorta.

(*d*) Pulsating perihepatic abscess

4. *Pulsations in the left axillary region* may be caused by:

(*a*) Enlargement of the heart, displacing the apex beat.

(*b*) Pulsating empyema.

(*c*) Aneurysm of the aortic arch.

(*d*) Chronic disease of the left lung and pleura, associated with retraction, thus exposing the heart's action more directly to the chest wall.

5. *Pulsation of the suprasternal notch* may be caused by:

(*a*) A dilated aortic arch (chronic aortitis) or subclavian arteries.

(*b*) An aneurysm of the aorta or subclavian

(*c*) A tumor or enlarged gland (thyroid and thymus) resting upon the transverse arch of the aorta which extends upwards into the neck.

6 *Systolic Retraction*· In thin individuals the systole of the heart usually causes a heaving impulse over the third, fourth and fifth interspaces on the left side, in line with the apex beat. A rhythmical retraction or sinking in of that region is significant of adhesive pericarditis.

7. *Broadbent's Sign*· A systolic retraction of the tenth and eleventh interspaces, below the inferior angle of the scapula, is in thin individuals occasionally symptomatic of pericardial adhesions The retraction is the result of a drawing upon the diaphragm by an hypertrophied and vigorously acting heart. This phenomenon may also at times be seen in cases of marked cardiac hypertrophy not associated with pericardial adhesions

C. The Apex Beat

It is of the greatest importance to study the apical impulse carefully. This impulse—generally spoken of as the *apex beat*—is the anatomical starting point for the further clinical study of the heart.

The apex beat, visible upon the chest wall of a healthy individual, does not represent the true anatomical apex or tip of the left ventricle As a rule, the impulse is caused by the tip of the right ventricle, which lies in contact with the anterior chest wall, and may be considered the *clinical* apex. The apex of the

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(*c*) Adherent pericardium; in this instance the chest wall is prevented from expanding because of adhesions between the pericardium and the parietal pleura: disuse of the intercostal muscles may result in slight atrophy, thus causing the general contour of the chest to be lost, and will produce a depression.

B. Precordial Pulsations (Other Than the Apex Beat)

1. *Pulsations at the base of the heart* may be caused by:

(*a*) Hypertrophy of one or both auricles.

(*b*) Retraction of the lung or pulmonary cavity in that part of the lung which covers the auricles.

(*c*) Aneurysm of the arch of the aorta

(*d*) Mediastinal tumor in close proximity to the aorta.

(*e*) Diffuse pulsation over the entire heart area, often seen in individuals with very thin and emaciated chest walls.

2. *Epigastric pulsation* may be caused by:

(*a*) Rapid heart action from any cause.

(*b*) Dilated right ventricle resting upon the diaphragm. The exaggerated impulse of the heart is transmitted to the diaphragm because of its close proximity. The diaphragm in turn transmits this impulse to a portion of the anterior abdominal wall, the epigastrium.

(*c*) Pulsating liver (*i. e.*, tricuspid regurgitation).

(*d*) Pulsating aorta, often seen in neurotic individuals with a thin belly wall

(*e*) Aneurysm of the abdominal aorta

Pathologic Displacement or Dislocation of the Apex Beat: The pathologic causes for displacement of the apical impulse may be summed up as follows:

I. *Cardiac Conditions:* Enlargement and dislocation of the heart.

II. *Extracardiac Conditions* Deformity of the thorax, pleural effusions (serous, purulent, sanguineous or gaseous), emphysematous lungs, pleural adhesions, shrinkage of the lungs, elevation of the diaphragm, mediastinal tumors, pericardial effusions

The apex beat may be displaced: (1) Upward, and to the left; or (2) to the right; (3) downward, and to the left; or (4) to the right; (5) to the left, and (6) to the right

1. *Displacement upward and to the left* may be caused by:

(*a*) Pericardial effusion: The heart, being an airtight hollow, muscular organ, will naturally float upon the surface of fluid. An effusion in the pericardial sac will, therefore, raise the heart upward, and at the same time rotate the apex toward the left. The apical impulse may be seen (when the patient leans forward) in the third or fourth interspace, close to the left anterior axillary line In the presence of pericardial adhesions the apex beat may be displaced downwards by a pericardial effusion

(*b*) Ascites, meteorism, large abdominal tumors, pleurodiaphragmatic adhesions and pregnancy will cause upward displacement of the heart to about the fourth interspace, and only slightly to the left of its normal position. The upward displacement in these cases is caused by the elevation of the diaphragm; it is easily differentiated from a pericardial effusion because in this condition the apical impulse is quite strong and is not influenced by posture, while in pericardial effusion the prone position almost entirely obliterates the apical impulse, because of the fluid gravitating toward the anterior chest wall, thus pushing the heart away from it.

(*c*) Upward traction upon the heart by retracted fibroid lung.

(*d*) Scoliotic or kyphotic deformity of the chest.

2. *Displacement upward and to the right* may be caused by:

(*a*) Conditions in the left chest which *push* the heart upwards and to the right, *i. e.*, a left-sided effusion, liquid or air, aneurysm of the lower part of the thoracic aorta occupying the left lower chest, or a large tumor occupying the left lower chest; also by abdominal conditions which so encroach upon the lower left chest as to push its viscera upward and to the right, *i. e.*, a greatly dilated cardiac end of the stomach or a diaphragmatic hernia, evisceration and eventration.

(*b*) Conditions which *pull* the heart upward and to the right, *i. e.*, fibroid phthisis of the right lung exerting an upward pull, or right-sided pleuropericardial adhesions pulling in an upward direction. The amount of displacement depends upon the quantity of displacing material in the left chest or the force of the pull on the right side; the greater the push or pull the more pronounced will be the displacement.

3. *Displacement downward and to the left* is noted in:

(*a*) Hypertrophy and dilatation of both ventricles. Hypertrophy of the left ventricle causes the greatest displacement downward and to the left, while hypertrophy of the right ventricle causes a greater displacement laterally. Simple downward displacement may be caused

left ventricle, or *anatomical* apex, extends further downward and toward the left, and is separated from the chest wall by a tonguelike projection of the lower lobe of the left lung. Only in great cardiac hypertrophy can the left ventricle produce a visible impulse.

The apex beat or impulse is usually seen as a regular, rhythmical systolic

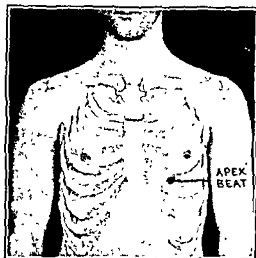


Fig. 11—Normal position of the apex beat, fifth intercostal space just beyond left parasternal line.

thrust, visible over an area of about one square inch. It occurs synchronously with the carotid and radial pulse, and is visible in the fifth interspace about one inch (2.5 cm.) to the right of the left midclavicular line, or about 3 or 3½ inches (7 to 9 cm.) to the left of the midsternal line.

The apex beat is studied by inspection, as to its (I) position, (II) extent, (III) strength, and (IV) rhythm

I. Position of the Apex Beat: In young normal adults, either in the recumbent or erect position, the apex beat is in the fifth interspace, just beyond the left parasternal line; or, as already mentioned, one inch (2.5 cm.) to the right of the left midclavicular line, or a little over 3 inches (7 cm.) to 3½ inches (9 cm.) to the left of the midsternal line.

Normal Variations: The apex beat may be displaced to a certain extent, and still be considered normal, *viz.*, in children up to the age of 10 years it is generally found behind the fifth rib, or in the fourth intercostal space in the mammillary line, or just outside of it. In old age, on the contrary, the apex beat is sometimes found in the sixth interspace, and nearer the median line. Persons having long narrow chests often have a visible cardiac apical impulse in the sixth interspace, while those possessing short broad chests may have their apical impulse in the fourth intercostal space. The difference in the location of the apical impulse in these two extremes is not so much because of the actual position of the heart, as on account of the slope of the rib.

Postural Change: When a person lies upon his left side the apex of the heart may shift an inch or more toward the left axillary line; a similar displacement to the right is observed, but to a less extent, when a person lies upon his right side. These alterations in the position of the heart on change of posture are caused by gravity, the heart's apex being lowered on the side upon which the patient rests.

Respiratory Change: The position of the apical impulse is little changed during quiet breathing, but during forcible inspiration, as the diaphragm sinks and the lower ribs are elevated, the apical impulse is carried downward and toward the median line. During forced expiration, it is carried upward and toward the left. In some instances a change amounting to the extent of an interspace, may be noted. A hyperdistended stomach will displace the apex beat upward.

tremely thick chest wall, likewise in those having very large lungs. If the apex chances to be behind a rib, the apical impulse may not be visible.

Pathologic Causes of Increase in the Apical Impulse: Any condition that increases the force of the heart, and, as often happens, its rate as well, will increase the area of apical impulse. For example:

1. Hypertrophy of the heart caused either by overwork or an endocardial lesion.
2. Dilatation with a certain degree of hypertrophy.
3. Nervous palpitation and excitement.
4. Exophthalmic goiter.
5. Drug poisoning (digitalis, alcohol, tobacco, tea, coffee and strychnine).
6. Retraction of the left lung (relative increase).

Pathologic Causes of Diminution or Absence of the Visible Apex Beat are:

1. Myocardial weakness from any cause as seen in chronic wasting diseases; prolonged acute febrile diseases; in shock, and after severe hemorrhage. If, throughout the course of a prolonged illness, the patient has had a fairly strong apical impulse, its disappearance may be regarded as indicative of grave danger.
2. Myocardial degeneration (fatty or fibroid).
3. Dilatation of left and right ventricles, with failure of compensation.
4. Overlapping of an emphysematous lung.
5. Pericardial adhesions.
6. Pericardial and pleural effusion.
7. Edema of the chest wall.
8. Inflammatory conditions of the cellular tissue of the left chest.

III. The Strength of the Apical Impulse: The strength of the apical impulse cannot be determined exclusively by inspection, but requires the aid of palpation A strong impulse at the apex is caused by hypertrophy of the left ventricle, hypertrophy of both ventricles, cardiac excitement, drugs or psychic influences, or a thin chest wall. As a rule, the strength of the apical impulse bears a direct relation to its extent, but it is often difficult to separate the apex beat from the "heartbeat" in general. There are, however, some cases in which there is an increase of force but not of extent.

By a "heaving impulse" is meant an apex beat which is so strong as to cause a distinct thrust upward of that portion *of the chest wall overlying the apex.*

A diminished or weakened cardiac impulse is due to dilatation of the ventricles, myocardial degeneration, pericarditis, adhesions and a thick chest wall.

IV. Cardiac Rhythm: Normally, the apex beat occurs at fixed intervals, with a given strength and rapidity, one beat being as strong as another, and each occurring after a pause of definite length This regularity is termed *normal rhythm* Pathologically, normal rhythm may be disturbed in the following ways:

1. Rapid heart action (tachycardia).
2. *Slow heart action (bradycardia).*
3. Irregular heart action (arrhythmia).

In (1) and (2) the heartbeats occur more or less frequently than the normal rate, but still they retain a certain amount of rhythm, because one beat is as strong or as weak as the other, and the intervals between the beats are of uniform length.

In *true cardiac arrhythmia* one impulse may be stronger than another, or the impulses may take place at irregular

by thoracic deformity, marked emphysema, aneurysm of the aortic arch, and by mediastinal growths pushing the heart downward; also by an enlarged liver pulling upon the central tendon of the diaphragm; and, to a lesser extent, by a moderate-sized, right-sided pleural effusion, or a pyopneumothorax.

4. *Displacement downward and to the right* may be caused by:

(*a*) Pleural effusion *pushing* a hypertrophied heart to the right, a mediastinal tumor or aneurysm exerting downward and inward *pressure* upon the left auricle, pericardial adhesions to the central tendon or right half of the diaphragm, and right-sided pleuropericardial adhesions *pulling* the heart downward and to the right.

5. *Displacement to the left* is noted in:

(*a*) Hypertrophy and dilatation of the heart (downward and outward).

(*b*) Pericardial effusion (upward and to the left).

(*c*) Right-sided pleural effusions, or pneumothorax, pushing the heart to the left.

(*d*) Pleuropericardial adhesions on the left side, pulling the heart toward the point of adhesion.

(*e*) Contraction of the left lung (apparent displacement).

(*f*) Hypertrophy and dilatation of the left ventricle.

6. *Displacement to the right* is noted in:

(*a*) Left-sided pleural effusion. Diaphragmatic hernia, eventration and evisceration, if left-sided, may push the apex beat behind the sternum, and in some instances, even as far as the right parasternal or midclavicular lines. The degree of displacement usually depends upon the amount of effusion and the mobility of the cardiac apex.

(*b*) Right-sided adhesive pleurisy with contraction—pulling the heart over.

(*c*) Transposition of the viscera (congenital); the heart is found in the right half of the chest instead of in the left; the position of the apex beat on the right side corresponds to its normal position on the left; *i. e.*, the fifth interspace beyond the parasternal line.

(*d*) Chest deformities because of disturbed anatomic relations may displace the beat in any direction.

Résumé of the principal causes of displaced apex beat:

1. *Hypertrophy and dilatation of the heart,* down and to the left.
2. *Pericardial effusion,* up and to the left.
3. *Chronic pleural and phthisical affections,* right or left.
4. *Emphysema* down and, sometimes, to the right.
5. *Pressure of subdiaphragmatic conditions,* up and, sometimes, to the left.
6 *Pressure of aneurysm or mediastinal growth* up and, sometimes, to the left.
7. *Chest deformities,* displacement in any direction

II. Extent of the Apical Impulse: The extent of the normal apical impulse in an adult, not too fat, is about 2.5 sq. cm. (one square inch). However, the normal apical impulse may vary in extent but an impulse greater than that usually is due to some pathological cause.

Normal Variation: The impulse may be *increased* in persons having thin chest walls, also after exertion and excitement, mental or physical, and after the ingestion of certain drugs, such as strychnine, alcohol and digitalis; it may be *diminished* or absent in normal persons who are very stout or possessed of an ex-

the temporal, facial or carotid arteries are selected.

The patient should be put entirely at ease before the examiner attempts to feel and count the pulse. The forearm should rest semipronated, either on the bed (if the patient is in bed) on the desk (in an office patient), or the forearm may be supported by the physician's free hand. The tips of the examiner's first three fingers are placed upon the radial artery in such a manner that the index finger rests farthest from the patient's heart, the examiner's thumb supporting the patient's wrist The three palpating fingers should ride gently over the artery in order to determine its texture. The pulse is then counted for one minute by the watch (all three fingers resting upon the artery). It is always better to "take" the pulse for a full minute than for a fraction of that period, as the information thus obtained is more reliable. In this way the rate, regularity and volume of the pulse wave can be accurately determined



Fig 14—Technic for taking the radial pulse

The next step is to note the degree of compressibility of the artery and the blood tension. The examiner's ring finger and middle finger are pressed at first gently, then firmly, against the pulsating artery, and the effect upon the compressibility of the pulse is noted with the index finger. This procedure helps to determine whether the pulse is easily compressible, moderately so, or wholly incompressible. It should be borne in mind that incompressibility of the pulse may be due either to hardening of the vessel wall, or to increased tension within the vessel. If caused by rigidity of the vessel wall (arteriosclerosis), the artery can be felt beyond the point of compression as a rigid cord, but when the noncompressibility is caused by high tension, no artery is felt beyond the point of compression, nor can the artery be felt at all during the diastole.



Fig. 15—Comparing both radial pulses

The last step, but by no means the least, is to palpate both radial arteries simultaneously, in order to determine their equality, frequency (rate), volume and rhythm.

the third or the fourth interspace, and does not always accompany each contraction of the heart. A pericardial friction rub may often be perceived as a to-and-fro friction sensation corresponding to the systole and diastole, for one or two minutes, then disappearing for a few minutes, only to reappear later. This fremitus may be brought out more plainly by moderate pressure with the hand over the cardiac area while the patient leans forward.

Pleuropericardial friction fremitus is perceived when the inflammatory condition occurs between the pericardium and the pulmonary pleura, its most common sites being the lingula of the lung, and at either side of the sternum where the pleural sac overlaps the pericardium. Pleuropericardial friction fremitus is recognized as a to-and-fro grating sensation, occurring during both the heart's action and respiration; "holding one's breath" will eliminate one source of the fremitus. Often it is difficult to differentiate between a thrill and a friction rub. The following table may be of assistance in making this differentiation

Thrill	Friction Fremitus
Harsh and vibratory in quality.	Grating, roughened, rubbing sensation
Conveys a sensation as if it came from the interior of the heart	Superficial quality.
Is not influenced by pressure, or respiration	May be influenced by pressure, posture and respiration.
Occurs over a valve.	Occurs over the body of the heart, or near the sternal edges.
Systolic, diastolic or presystolic in time, depending upon its cause.	To-and-fro in time.

Valve Shock: This may be felt as a result of an accentuated closure of one or more valves. It may often be palpated in thin persons who have hypertrophied or rapidly acting hearts, or in persons who present some resistance to the blood current. If a valve shock is felt over the pulmonary orifice, it indicates increased resistance to the pulmonary circulation; if it is felt over the aortic orifice, it indicates systemic engorgement. Valve shock is analogous to accentuation of a certain valve sound; it should not be mistaken for a thrill, a mistake not infrequently made by the beginner.

The Pulse

By *the pulse* is meant the wavy impulse of an artery as a result of its expansion and contraction; it is transmitted to the finger tips while palpating a superficial artery. The expansion is due to the momentary increase of blood pressure in the arterial tree produced by ventricular systole. The pulse wave causes a change in the shape of the artery, *i. e.*, from an oval to a circle.

Technic for Taking the Pulse: Any superficial artery that is easily accessible to the finger tips may be selected, the only requisite being that the vessel so selected may be compressed between the examining finger and a firm point, such as a bony prominence. The radial artery is usually preferred because it is readily accessible, and is not easily influenced by disease of the structures it supplies Under some circumstances, when it is most convenient, as during anesthesia, or where the radial artery is not palpable.

wave is sometimes found in cases of severe asthenia, where the arteries have lost their muscle tone, so that each ventricular systole causes a hyperdistension of the artery. Such a pulse is easily compressible.

Corrigan's or *water-hammer pulse* or *trip-hammer pulse* is an abnormally full and not easily compressible pulse, which collapses suddenly when its height is reached. This is found in aortic regurgitation.

Pulsus vacuus (empty pulse) or *pulsus parvus* (small pulse): A *small* pulse, if not caused by abnormally small arteries, is also an *empty* pulse, and is due to diminished work of the heart, particularly of the left ventricle, as is seen in mitral stenosis, and in the combined lesions of aortic stenosis and mitral regurgitation Partial obstruction of an artery will, for obvious reasons, cause a small pulse, as will also severe anemia, profuse hemorrhage and myocarditis.

Thready or *filiform pulse* is a very small and empty pulse, while *pulsus tremulus* (trembling pulse) is a very small, but nevertheless full, pulse. These two conditions are found when the heart is extremely weak (myocarditis). *Wiry pulse* is a small noncompressible pulse, usually very fast, seen in scarlet fever

Dicrotic pulse is a soft pulse having a double impulse; the second or smaller impulse is caused by the rebound of the pulse wave. This type of pulse is found in exhausting febrile conditions, typhoid, etc. In order to demonstrate this pulse the patient's elbow must rest upon some object (bed), the forearm being at right angles with the arm, and the fingers pointing upward.

III. **Rhythm or Regularity:** The rhythm of the pulse may be disturbed in two ways: (1) Arrhythmia as to time (pulse throbs do not follow one another at regular intervals); (2) arrhythmia as to volume (regular as to time, but variable as to volume). Often there exists a combination of (1) and (2), as the irregular pulse may be unequal in volume.

1. *Arrhythmia as to Time:* A slight degree of irregularity as to time may be encountered in persons who show no other evidence of disease. A regular intermission occurs at a given number of beats and corresponds to a similar phenomenon in the heart. If the pulse is normal in all other respects, this phenomenon may be considered as an individual peculiarity, the cause of which is attributed to *ventricular extrasystole.* An irregular pulse may occur temporarily in emotional excitement, fatigue, neurasthenia, because of overindulgence in tobacco, tea and coffee, and in constipation and various digestive disorders; it is also seen at times in the very young and in the aged as a result of *sinus arrhythmia* (SEE: pp. 439 and 518).

Persistent arrhythmia, associated with the signs of circulatory disturbance, is a grave condition, and may be due to disease of the heart muscle, disease of the nervous mechanism of the heart, or to reflex causes Absence of rhythm usually occurs after failure of compensation, though in mitral stenosis arrhythmia may occur long before other signs of ruptured compensation are detected It usually indicates *auricular fibrillation*

The abuse (the use of too large doses, or too long continued administration) of digitalis in cardiac diseases, may cause arrhythmia (coupling or slowing of beats) until the drug is withdrawn

Pulsus bigeminus is a pulse in which the beats run in pairs; each pair is separated by a prolonged pause.

10. The various arrhythmias—auricular flutter, auricular fibrillation—and all forms of tachycardia, whether idiopathic or otherwise, show a very rapid pulse rate. The pulse is also increased in:

11. Anemia, all forms.

12. Debility and Addison's disease.

13. Excessive use of tobacco or alcohol, sexual excess, lack of sleep.

14. After hemorrhage; after aspiration of a pleural exudate; in the presence of ascites, and during convalescence from acute diseases.

15. Aneurysm, pleural effusion and empyema.

16. Distention of the abdomen, peritonitis or tympanites, and enlargement of certain abdominal organs, *i. e.*, spleen, liver and kidneys

17. The use of drugs — atropine, strychnine, alcohol, caffeine, suprarenal extract, coal-tar derivatives

Diminished Frequency (bradycardia): In some individuals the pulse rate is normally slow, often being no faster than 40 to 60 per minute. In the aged the pulse may be only 60 or less per minute

Physiologically, its rate is lessened during sleep, absolute rest, the puerperium, or convalescence from certain fevers (typhoid, pneumonia, etc.).

Pathologically, the pulse may be slow in:

1. Myocarditis.

2. Myxedema, in the early stages.

3. Meningitis, typhoid fever, vagus irritation, arteriosclerosis.

4. Intracranial pressure by tumor, hemorrhage, edema, effusion, etc.

5. In certain forms of mania.

6. Melancholia and hysteria

7. After poisoning by drugs, such as opium or digitalis

8. In toxemia due to absorption of bile and urea.

9. In epilepsy, a pulse which becomes slow after having been rapid for a long time, should be regarded as a danger signal.

10. A slow or infrequent pulse occurring in cardiac diseases indicates fatty degeneration of the heart muscle, and probably, disease of the coronary arteries. A slow pulse may at times occur in the presence of a rapidly acting heart because all the impulses are not transmitted to the radial artery (pulse deficit). This is often seen in certain types of arrhythmia (auricular fibrillation).

11. Stokes-Adams' syndrome, that is, bradycardia with epileptoid or syncopal attacks, may occur when the pulse rate drops to from 15 to 25 per minute.

12. The various forms of heart block.

II. **Force or Quality and Size of the Pulse**: By the quality of the pulse is meant the size of the pulse wave and its degree of tension. There are so many variations in the quality of the normal pulse that it requires a great deal of experience and diligent practice to recognize pathologic changes

The size of the pulse depends upon the amount of blood thrown into the circulation by each cardiac systole, and upon the size and position of the artery palpated. Thus, persons who have naturally large arteries will show a larger pulse than those who have small superficial arteries; or again, the radial artery may run an anomalous course, thereby making proper deductions difficult

Pulsus plenus (full pulse) or *pulsus magnus* (large pulse) is found in conditions of plethora and in hypertrophy of the left ventricle, providing such hypertrophy is not caused by a serious valvular defect. A large broad pulse

wave is sometimes found in cases of severe asthenia, where the arteries have lost their muscle tone, so that each ventricular systole causes a hyperdistension of the artery. Such a pulse is easily compressible.

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The abuse (the use of too large doses, or too long continued administration) of digitalis in cardiac diseases, may cause arrhythmia (coupling or slowing of beats) until the drug is withdrawn.

Pulsus bigeminus is a pulse in which the beats run in pairs; each pair is separated by a prolonged pause.

Pulsus trigeminus is one in which every third beat is followed by a pause.

2. ***Arrhythmia as to Volume:*** *Pulsus alternans* is characterized by the regular alteration of a small feeble pulsation with one that is larger and stronger; that is, the pulse is regular in rhythm but irregular in volume. This condition is found in advanced myocarditis and is a grave prognostic omen.

Pulsus myurous (rare) is a peculiar condition described by older writers. A full and forcible pulse wave is followed by a series of several beats gradually decreasing in volume, this succession of changes being maintained with a certain degree of regularity (also called decurtate or mouse-tail pulse, seen during Cheyne-Stokes respiration).

Other Irregularities: *Pulsus intercidens* is characterized by the occurrence of a small or rudimentary extra beat after several perfectly normal pulse beats (seen in extra systoles).

Pulsus Paradoxus The "paradoxical pulse" of Kussmaul is characterized by the disappearance of the pulse wave with each deep inspiration. It is said to be due to adhesive pericarditis, pericardial effusions, mediastinal inflammation, or to tumors or adhesive bands compressing the aorta during deep inspiration.

Intermittent pulse is characterized by the dropping of two or more impulses after several regular pulse waves have occurred. This is caused either by the periodic interruption of the heart's action, or by insufficient power of the heart muscle to cause a radial impulse

Pulsus deficiens occurs when the dropped pulse waves are caused by periodic rudimentary heartbeats which are not of sufficient strength to be registered at the radial artery.

Irregular intermittent pulse is a pulse which is irregular in its irregularity, no two beats or cycles being alike. It is irregular as to *time, volume, rhythm,* and *force;* in fact, it lacks practically all the attributes of a normal pulse. This variety of pulse is often met with in severe cases of auricular fibrillation.

Pulse rhythm may also be studied by the sphygmograph.

IV. Condition of the Arterial Wall: An artery that feels round and is not easily compressible may indicate increased blood tension within the artery, or sclerosis of the artery wall. If the artery cannot be felt beyond the point of compression, the increased tension is caused by increased blood pressure. Often the two conditions, increased arterial tension and sclerotic arteries, coexist. An artery that has undergone marked sclerotic changes is usually recognized by the following points:

The artery is longer than normal, therefore, it becomes tortuous. It feels hard and round, and is easily rolled under the finger. Beyond the point of compression, the artery can be felt like a whipcord and is often beady. The diastole, or period between pulse waves, produces very little change in the size and shape of the vessel.

V. Tension: Arterial tension depends upon five distinct conditions:

1. *The amount of blood in the circulation.* The more blood the higher the tension. Also the viscosity of the blood has a direct bearing on the tension.

2. *The size and vigor of the left ventricle.* A strong hypertrophied left ventricle will produce a high tension pulse; a degenerated left ventricle will produce a low tension pulse.

3. *The condition of the arterioles.* Increased resistance in the arterioles will cause a high tension pulse.

4. *The condition of those organs which receive a supply of arterial blood.* If the organs are congested or fibrotic, the tension will be high.

5. *The condition of the glands of internal secretion.* Some of the endocrine glands and the sympathetic nervous system seem to have a definite influence upon arterial tension.

Blood Pressure

The finger is a poor indicator of the degree of tension in the artery. In most cases palpation of the artery will reveal either an increased or decreased tension; seldom, however, can even the most experienced observer tell the actual amount of pressure with any degree of accuracy. To gauge accurately the tension, the sphygmomanometer, an instrument devised for accurately determining the blood pressure during systole and diastole, is employed.

Systolic Pressure: By systolic pressure is meant the amount of pressure exerted upon the caliber of the arteries during the systole of the heart; it is measured by the number of millimeters of mercury required to compress the radial artery.

Diastolic Pressure: By diastolic pressure is meant the amount of blood pressure constantly present in the vessels during the diastole of the heart.

The pulse pressure is obtained by subtracting the diastolic from the systolic pressure; this represents the force exerted by each systole Thus, if systolic pressure equals 120 and diastolic pressure equals 80, pulse pressure will equal 40 (120—80=40).

The mean pressure is obtained by adding the systolic pressure to the diastolic and then dividing by 2 Thus, if systolic pressure equals 120 and diastolic pressure equals 80, the mean pressure will equal 100 (120+80=200÷2=100).

Since the introduction of the sphygmomanometer the estimation of "blood pressure" has practically become an accurate science, and a physician can no more afford to be without a blood pressure instrument than without a clinical thermometer.

Hypertension and Hypotension

Alteration in arterial tension should not be regarded as a distinct pathological entity, but only as a symptom of dysfunction This is true, irrespective of whether the etiologic factors are or are not apparent Exceptions may be made in the case of certain clans or families whose members uniformly present a somewhat higher or lower blood pressure.

Etiology: The precise mechanism operative in the deviation of blood pressure, either above or below the arbitrary normal, is as yet not entirely explainable It is, however, known that certain pathologic states have a definite effect upon arterial tension; also that hypertension or hypotension may occur in individuals who in other respects seem to be perfectly normal It is quite feasible that arterial tension may be controlled by a not as yet identified "center" in the brain, in the adrenals, in the medulla or in the kidneys

Hypertension: This may be defined as an increase of the systolic and diastolic arterial blood pressure with or without an increase of the pulse pressure Blood pressure above 150 systolic and 90 diastolic in persons below 50

years of age, and 160 systolic and 90 diastolic in persons past 50 years of age may be considered above normal. Pressure of 260 to 300 systolic and 120 to 140 diastolic may be found in individuals presenting no other abnormality (*essential hypertension*), though with the lapse of time such persons will show definite evidence of disease in the blood vessels of the brain, heart or kidneys, because no one is so constituted as to bear such a terrific strain without giving way at some point.

In the following conditions high blood pressure is a prominent symptom:

Nephritis of the glomerular type with nitrogen retention; urinary obstruction

Arteriosclerosis with hypertension and cardiac hypertrophy (SEE p. 525)

Chronic intestinal toxemia, toxemia of pregnancy.

Chronic focal infections

Aortic insufficiency (high systolic and low diastolic).

Sclerosis of the cerebral vessels.

Cerebral hemorrhage.

Increased intracranial tension.

Obesity; polycythemia; pituitary basophilism.

High tension living, constant excitement and anxiety.

Endocrine disturbance as seen in women at the menopause and in hyperadrenalism, hyperpituitarism and hyperthyroidism.

Sympathicotonia.

Hypertrophy of the prostate gland is often associated with hypertension which is frequently attributed to age and arteriosclerosis However, the removal of a pathologic prostate may permanently relieve the hypertension.

Essential Hypertension (Hyperpiesis, Primary Arterial Hypertonia): Essential hypertension during the early stage acts as a functional disturbance of the vasomotor system, showing no abnormalities other than an increase of the systolic and diastolic pressure above the accepted normal. As the disease progresses there develop cardiac hypertrophy, increased arterial tonicity, spasticity of the retinal arteries with tortuosity of the retinal veins. During the late stages, there may develop severe symptoms referable to the cardiovascular system, the brain or the kidneys. The disease may affect equally the entire arteriolar system, or one group of vessels may bear the greatest brunt. The symptoms depend upon the stage of the disease, and the amount of pathology in the organs chiefly involved.

General Symptoms and Clinical Findings: During the early or benign stage, aside from a moderately elevated systolic and diastolic pressure, there may be no symptoms. When subjective symptoms do appear, those most frequently found are headache, vertigo, ringing in the ears, irritability and heart consciousness. Excitement aggravates these complaints and raises the tension. During the later stages there may develop pathologic manifestation in the cardiovascular system, the brain or the kidneys.

The Cardiovascular Manifestations: The walls of the arteries and arterioles become thickened and their lumina narrowed. This leads to cardiac hypertrophy. When the hypertrophy becomes massive, there develops coronary insufficiency with reduction of blood flow, and this leads to cardiac ischemia and anoxemia, thus resulting in myocardial failure. Hypertensive heart failure is a frequent cause of death in persons above the age of 55 years (SEE p 493).

Cerebral Manifestations Cerebral vascular spasm is fairly common This causes transient cerebral symptoms such as paresthesias, motor or sensory aphasia, monoplegia, hemiplegia, epileptiform seizures, local twitchings, severe headache, vertigo and, at times, temporary

blindness. Eventually there may develop hypertensive encephalopathy, thrombosis or hemorrhage. The latter two conditions are among the frequent causes of death in essential hypertension Cerebral hemorrhage occurs more frequently in the region of the basal ganglia.

Renal Manifestations· In this disease nephritis is not the cause of the hypertension. It is the hypertension associated with arteriolar hypertrophy and fibrosis which limits the blood supply to the kidneys and causes the primary contracted red granular kidney, so common in this disease when the kidneys are involved

Malignant nephrosclerosis or malignant *hypertension is a severe stage of* hypertension in which the kidneys bear the greatest brunt of the disease. It usually occurs in comparatively young persons. The blood pressure is exceedingly high, 250 to 300 systolic and 120 to 160 diastolic, and kidney function is poor; retinal sclerosis is nearly always present, while retinal hemorrhage and choked discs are not frequent findings. Essential hypertension usually runs a protracted course, but when the stage of malignant hypertension is reached, death may occur in a comparatively short time from uremia or vascular crisis.

Etiology The cause of essential hypertension is as yet not definitely proven It is believed by Goldblatt to be due to a pressure substance secreted by an ischemic kidney There are also other theories but none are proven The disease has a familial tendency

Diagnosis Before a diagnosis of essential hypertension is made one must exclude the known conditions that cause high blood pressure (SEE: pp. 412 and 525). A systolic pressure persistently above 160 and a diastolic pressure above 90 associated with spasticity of the retinal vessels, even in the absence of any other abnormal manifestations, may be considered as essential hypertension in a benign or early stage.

Hypotension: This may be defined as a decrease of the systolic and diastolic arterial blood pressure. Values below 90 systolic and 50 diastolic may be considered pathologic. Constant low blood pressure is often a familial characteristic and is consistent with longevity. "Low pressured" individuals may fatigue easily, but often after a brief rest continue with their tasks and in the end outdistance the "high pressured" individual. Pathologically low blood pressure may be caused by:

- Severe asthenia
- Pulmonary tuberculosis
- Addison's disease.
- Cardiovascular degeneration, mitral and aortic stenosis
- Hypopituitarism, hypothyroidism
- Coronary thrombosis
- Arteriosclerosis associated with cardiac degeneration
- Vasomotor disturbance.
- Vagotonia
- Shock.
- Severe anemia, severe hemorrhage.
- Prolonged febrile conditions.
- *Lipoid nephrosis*

Hypotension following hypertension is often of grave prognostic omen.

Coronary thrombosis in the hypotensive individual is often more serious than in the hypertensive individual.

Pulse Pressure: The pulse pressure may be high because of an increase of the systolic pressure without any corresponding increase of the diastolic pressure. This is often seen in nervous hypertension, or temporary hypertension due to stimulation, excitement, or mental and physical exertion. The pulse pressure may also be high because of a drop in the diastolic pressure as seen

years of age, and 160 systolic and 90 diastolic in persons past 50 years of age may be considered above normal. Pressure of 260 to 300 systolic and 120 to 140 diastolic may be found in individuals presenting no other abnormality (*essential hypertension*), though with the lapse of time such persons will show definite evidence of disease in the blood vessels of the brain, heart or kidneys, because no one is so constituted as to bear such a terrific strain without giving way at some point.

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where the loud booming sound changes suddenly to a weak thud

Technic for the Auscultatory Method: The sphygmomanometer is adjusted as previously described. The chest piece of a binaural stethoscope is applied (without pressure) a little below the bend of the elbow, over the ulnar artery, the other end of the stethoscope being held in the examiner's ears The cuff is inflated beyond the point where the pulse sound is obliterated. The cuff is then slowly deflated until a distinct short beat is heard This is marked as the *systolic pressure.*

The cuff is further deflated, the sound marked as the systolic point undergoing a number of modifications; at first feeble, it soon changes to a broad murmur; this, in turn, gives place, as the pressure is released, to a strong clear cut, short sound, which is heard until it reaches a point where it suddenly becomes soft and indistinct This point is marked as the *diastolic pressure*

The five phases through which the auscultatory pulse sound passes are described as follows·

First phase represents the sound as first heard after complete compression; it indicates the systolic pressure and much resembles the apical heartbeat It is caused by the return of the pulse wave in the artery at a definite stage of compression.

Second Phase: The sound simulating the systolic heartbeat of the first phase becomes a *hissing murmur,* caused probably by the uneven constriction.

Third Phase: The sound is now clear cut, short and snappy; it becomes louder as the pressure is released, until a point is reached where it suddenly becomes weak; which point is recognized as the fourth phase.

Fourth Phase: The sudden transition from the third phase to the fourth gives one the impression of a boulder which, rolling along a level surface, suddenly drops over a precipice. This point marks the *diastolic pressure.*

Fifth phase is represented by the continuance of the weak sound until its final cessation It is evident that the sound in an artery depends upon the amount of constriction of that artery When the artery is entirely obliterated, there is no sound, as is also the case when the artery is not at all constricted The five phases just mentioned occur as a result of the degree of constriction of the brachial artery.

Percussion

It is of great importance for the student to practice cardiac percussion



Fig 16—Percussion of thorax in the extremely modest

with as much care and concentration as possible. Unless one has a sharp ear, proper technic and a uniform method of procedure, cardiac percussion will

in aortic regurgitation, after exertion in cardiovascular weakness, in exophthalmic goiter, in shock, in hemorrhage and, at times, in anemia. *A high pulse pressure* also occurs in general hypertension where both the *systolic and diastolic pressures* are increased, the systolic usually rising *out of proportion to the diastolic.*

A low pulse pressure usually occurs in arteriosclerosis with hypotension. The diastolic pressure is proportionately high in cardiac decompensation with cyanosis and edema, in coronary thrombosis, and in any condition where venous stasis is present. Whenever the systolic pressure falls below the pulse rate an unfavorable prognosis may be anticipated. The same holds true of any condition in which the diastolic pressure falls below the respiratory rate. The normal pulse pressure is usually between 40 and 50

Variation of Blood Pressure with Age and Sex: At birth the systolic pressure varies from 35 to 50 mm. Hg At the tenth year it is about 80 to 90 mm. Hg. At the sixteenth year the systolic pressure varies from 90 to 120 mm Hg In the adult, Rolleston's formula is 100 plus age. This formula is remarkable for its variations. The systolic pressure in women is usually 5 to 10 mm. Hg lower than in men. The diastolic pressure up to the fiftieth year is usually two-thirds of the systolic. In the aged, the *diastolic pressure may* be *one-half* of the systolic pressure.

Technic for "Taking" Blood Pressure: ***Step One:*** The patient should assume a *perfectly unconstrained* position, either lying in bed or sitting upon a *chair; all muscles should be relaxed as much as possible.* The arm nearest *the examiner should* be bared, or a very thin garment may be worn. The "cuff" of the sphygmomanometer is snugly wound around the arm, and the free end is fastened, so as to prevent loosening The two pieces of rubber tubing connected *with the cuff* are disposed of as follows:

The end of one tube (it does not matter which) is attached to an air bulb, while the other tube is attached to the sphygmomanometer. The instrument is now ready for use. Either the auscultatory, palpatory or the combined auscultatory and palpatory methods may be used.

Step Two (palpatory method): The examiner takes the radial pulse of the patient's constricted arm with whichever hand is most convenient. With the other hand he grasps the air bulb and slowly inflates the cuff until the radial pulse is entirely obliterated. It is best to go several degrees beyond that point and then gradually deflate the cuff until the pulse on its return becomes barely perceptible to the palpating fingers This point is then marked as the *systolic pressure.*

Step Three: The diastolic pressure is most accurately obtained by the *auscultatory method.* When the *palpatory method* is used, we depend chiefly upon observing the greatest oscillation of the column of mercury or the needle (*in* spring instruments) The "cuff" is gradually deflated, and when a point is reached at which the mercury or the needle shows the greatest oscillation, this point is marked as the *diastolic pressure* By the *auscultatory method* (which is the most accurate and therefore the method of choice) the systolic pressure is marked at the point of compression when the pulse sound is first heard after having been *obliterated* by the pressure of the inflated "cuff." The diastolic pressure is marked at the point

hand while the examiner taps the chest wall with the index or middle finger of the other hand. Tapping is also started in the resonant part of the chest, the heart being gradually approached Thus, the intercostal spaces and not the ribs, are percussed Cardiac dullness is best elicited by mapping out three points:



Fig 18—Technic for immediate (direct) heart percussion

1. *Upper Point.* Percussion is started from the left clavicle and carried downward and inward until dullness is reached

2. *Right Lower Point:* Percussion is started in the fourth intercostal space and midclavicular line, and carried inward until dullness is reached

3. *Left Lower Point* Percussion is started in the left eighth interspace and anterior axillary line, and carried upward and inward until dullness is reached. A line connecting the three points represents cardiac dullness.

Cardiac Dullness

We speak of two forms of cardiac dullness, *superficial* and *deep,* as follows:

1. *Superficial* (exposed, actual or absolute) cardiac dullness corresponds to that portion of the heart not covered by lung. The anterior portion of the right ventricle lying in the quadrilateral space, is in close contact with the chest wall It, therefore, requires only a superficial percussion stroke to bring out actual dullness This space is bounded: *Superiorly,* by the upper edge of the left fourth costal cartilage, in the parasternal line; the *right border* extends along the right edge of the sternum from its upper boundary to about the sixth rib where it blends with liver dullness; the *left border* corresponds to a curved line with its convexity outward, running just inside the parasternal line, and joining the upper area of cardiac dullness to that elicited at the sixth interspace.



Fig 19—Technic for outlining cardiac dullness by immediate percussion

The *lower border* of the heart cannot be outlined by ordinary percussion, because it blends with liver dullness, but it may often be determined by auscultatory percussion, or by the use of the tuning fork, by which methods it is often

yield no satisfactory results. The outline of the heart as obtained by percussion is somewhat smaller than actual size, as has been proven by radioscopy. The difference is no doubt due to lung resonance, encroaching upon cardiac dullness.

The object of percussion is to determine: (1) The size of the heart, actual, relative and exposed; (2) the position of the heart, and (3) the presence of enlargement of any one of its chambers.

Technic: The technic employed in the general percussion applies also to cardiac percussion.

The heart is an airless organ, and therefore, gives rise to a dull sound; it is surrounded on three sides (upper, right and left) by air-containing or resonant tissue. The transition from resonance to dullness marks the location of the borders of the heart. Percussion should always be started on the resonant tissue and the supposed outline of the heart approached in parallel lines along its various borders. The percussion stroke should be rather forcible.

Percussion of the heart, like that of the lungs, may be either *mediate* or *immediate.*

Mediate Percussion: The finger is the only medium used, as the employment of instruments for outlining the heart is impractical. The pleximeter finger is placed, if possible, in an interspace, only the distal phalanx being laid upon the chest wall, while the other parts of the finger are raised so as not to interfere with chest vibrations. The pleximeter finger is then struck sharply at the rate of two per second, with the soft part of the middle finger, nearest the nail. The border of the heart is approached in each interspace from the resonant area.



Fig 17—Technic for orthopercussion

Orthopercussion: This is practically a form of mediate percussion. The pleximeter finger is bent at the second joint and held at a right angle to the hand, the tip of the finger resting upon the chest wall The plexor finger strikes the pleximeter finger lightly upon the second phalanx. It is claimed by many physicians that the heart border is more easily outlined by this method.

Immediate Percussion: Of late this has become greatly in vogue, and is favored by many competent clinicians. The precordial skin is drawn taut with one

fourth rib. The total area of cardiac dullness is represented by a combination of the covered and exposed areas of cardiac dullness.

Area of Vascular Dullness: Percussion over the sternum elicits a peculiar bony resonance masking both cardiac dullness and lung resonance. The aorta and superior vena cava are situated behind the sternum above the second rib, and on deep or forcible percussion an impaired osseous resonance may be elicited over that area

Conditions Modifying the Normal Areas of Cardiac Dullness: Both areas may be proportionately increased or diminished, or the dimensions of one may be altered at the expense of the other. Normally, the size and location of the percussion areas are influenced by the:

(*a*) ***Age of the Individual:*** In children the lungs are relatively small, and the dull areas of the heart and liver correspondingly greater. Early in life, because of the greater elasticity of the lungs, the area of relative dullness is relatively increased during full inspiration, while that of actual dullness is diminished; a reversal of these conditions is obtained during shallow breathing and forcible expiration It should be remembered that the area of absolute dullness is greater in children than in adults, and that its upper limit is about one interspace higher; also that it extends a little

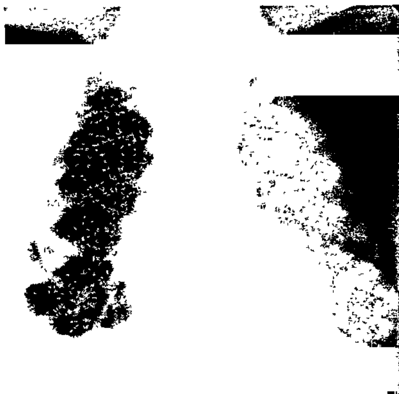


Fig 21—Case of aortic insufficiency—extreme enlargement of left ventricle due to hypertrophy and dilatation; note the stocking-shaped shadow.

possible to determine where the liver ends and the heart begins.

The *cardiohepatic angle* or *Ebstein's angle* is a right angle of resonance caused by the junction of the horizontal limit of hepatic dullness with the upright line of cardiac dullness, thus forming an approximate right angle of resonance in the fifth right intercostal space, close to the sternal border.

2. *Deep*, covered or relative area of cardiac dullness represents that portion of the heart covered by lung as outlined on the anterior surface of the chest, and can be demonstrated only by a forcible percussion stroke. The sound thus elicited is relative dullness, because the lung resonance blending with the cardiac dullness produces this modified sound of relative dullness or impaired resonance.

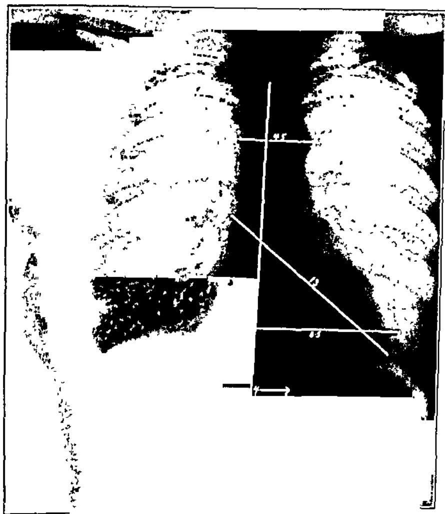


Fig 20—X-ray of normal heart and lung

The *boundaries* of relative cardiac dullness correspond to the outline of the heart, minus the exposed portion. The upper boundary is the third rib; the right boundary, a *little to the left of the* right parasternal line; the left boundary slightly to the right of the left midclavicular line, and the lower boundary at the

(*a*) *Hypertrophy and Dilatation of the Heart:* If the dullness extends to the right, it indicates right ventricular enlargement, and, at times, enlarged vena cava, or moderate pericardial effusion; the latter condition often obliterates Ebstein's angle If dullness can be detected to the left and downward, it means left ventricular enlargement, if to the right, and over the third and fourth interspaces, right auricular enlargement; if to the second left interspace, left auricular enlargement. Circumscribed dullness in the second interspace, close to the sternum, is often found in aortic stenosis, due, no doubt, to left auricular hypertrophy.

(*b*) *Pericardial effusion* gives rise to an enormous area of absolute cardiac dullness; it can be differentiated from hypertrophy of the heart by the following points:

Pericardial Effusion	Hypertrophied Heart
Large area of dullness and flatness; base downward and apex up	Dullness, base upward, apex downward
Change of outline of dullness on change of posture, without change in the position of the apical impulse	No greatly appreciable change
Relative dullness not obtained, the note changes from lung resonance to flatness because the pericardial sac is filled with fluid, which pushes the lungs away from the heart	Relative dullness gradually merging into actual dullness.
Apex beat displaced upward and to the left (as a rule)	Apex beat displaced downward and to the left, and changes in change of posture.
Cardiohepatic angle (Ebstein's angle) is obliterated	Cardiohepatic angle (Ebstein's angle) present.
Rotch's sign (dullness 1 to 2 inches to the right of the sternum) is positive.	Rotch's sign absent

Extrinsic causes or *apparent* increase in the area of heart dullness may be due to:

(*a*) Shrinkage of the lungs, thus exposing a greater portion of the heart.

(*b*) Consolidated lung near the heart simulating an increased area of heart dullness, there being no way of differentiating by percussion between a consolidated lung and the heart

(*c*) Tumors or enlarged glands encroaching upon the heart, causing extension of cardiac dullness

(*d*) Aneurysm of the ascending portion of the aortic arch, the dullness extending above the normal cardiac area, and to the right of the sternum. Extension of dullness over the manubrium may indicate aneurysm of the transverse portion of the same vessel Dullness to the left of the sternum, in the first or second interspaces, may indicate aneurysm of the descending portion at its beginning. Aneurysmal dullness does not displace the normal area of cardiac dullness, but is superimposed upon it Dullness over the upper part of the sternum may also be caused by a persistent thymus, substernal goiter or mediastinal neoplasm

II ***Diminished or Absent Cardiac Dullness:*** This may be caused by atrophy of the heart, although this is a rare condition. As a rule, diminished or absent cardiac dullness is due to some extrinsic cause, such as emphysema of the lungs The distended hyperresonant lung covering a greater part of the heart

farther over to the left, and does not descend to so low a level as in adults In old age, even in persons who are otherwise in good health, the borders of the lungs are usually emphysematous; hence, the area of superficial dullness is smaller. Relative dullness is also much lower, because the heart hangs lower in the thoracic cavity of the aged than in the young adult. The upper area of relative dullness may be found at about the lower border of the fourth rib, and the upper border of actual dullness may be as low as the fifth rib; the left border may also be nearer the median line.

(b) Position of the Patient: The percussion area of relative dullness is the same, whether the patient lies on his back or occupies the upright position. But the area of actual dullness is diminished when the patient lies on his right side, and is increased when lying on left side.

(c) Condition of the Lungs: The area of actual cardiac dullness is diminished during deep inspiration, and increased during full expiration.

(d) Position of the Diaphragm: When the diaphragm is raised, the heart dullness is higher, and when this muscle is depressed, cardiac dullness reaches a lower level.

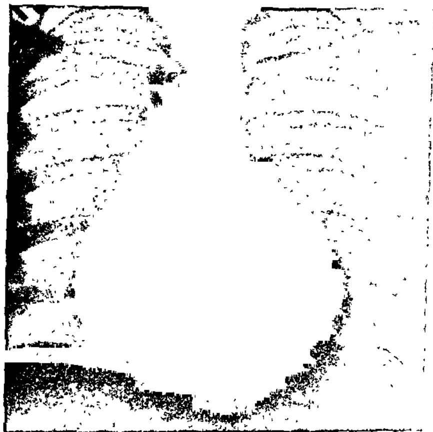


Fig. 22—Case of pericardial effusion of moderate degree; note the pear-shaped shadow

Abnormal Areas of Cardiac Dullness: Heart dullness may be increased, diminished, absent or misplaced.

I. *Increased Cardiac Dullness:* This may be due to *intrinsic* or *extrinsic* factors. It is *intrinsic* when due to some condition of the heart itself, such as:

The patient should remain in the same posture he assumed during percussion, though it may sometimes become necessary to have him lean forward, or he



Fig. 24—Auscultating the apex beat

upon his back, or upon his left side, or he may have to raise his hands above his head several times in succession, in order to bring out stronger heart sounds, or



Fig 25—Auscultating the apex beat

to note the effect of exercise and posture upon the cardiac sounds A murmur may become more audible after such a procedure, particularly if the heart sounds are weak because of degeneration of the heart muscle.

In ambulatory patients it is often necessary to have them walk across the floor, or run up and down a flight of stairs, or hop on one foot a number of times, the heart being auscultated both before and after the exertion. With the patient in proper position, the following areas are examined:

1. *Mitral Area* The stethoscope is placed over the apical area (fifth inter-



Fig 26—Auscultating the pulmonary valve

space near the nipple) so that the character of the heart sounds may be noted If the sounds seem normal, the second area is then auscultated, but if an adventitious sound is heard over the mitral area, the exact character and time should be noted, and the sound followed toward the left axilla to the angle of the left scapula

2. *Pulmonic Area* The second area of auscultation is in the second intercostal space at a point close to the left sternal line The character of the sound, the presence or absence of adventitious sounds, and the presence or absence of

than under normal conditions will encroach upon heart dullness, and, in extreme cases, it may entirely overlap the heart. A greatly distended stomach, particularly at its cardiac end, may cause a diminution of cardiac dullness because the gastric tympany will mask the dull sound normally elicited over the heart.

Pneumopericardium: Air in the pericardial sac is a rare condition, but when present will cause hyperresonance or tympany instead of dullness over the precordia.

Pneumothorax: Either spontaneous or artificially induced pneumothorax may cause diminished or absent cardiac dullness, depending upon its size and location.

Gastric Carcinoma: This condition reduces, and at times obliterates, absolute cardiac dullness in the recumbent posture (W. Gordon).

Displaced Cardiac Dullness: A displaced heart cannot be accurately outlined by percussion alone, because the cause of the displacement may often give rise to similar dullness, *i. e.*, pleural effusion, neoplasm, or aneurysm. By observing the apex beat, the outlines may at times be inferred by percussion. In cases of *dextrocardia* (*situs inversus viscerum*) the size of the heart may be outlined on the right half of the chest.

Auscultation

Auscultation of the heart is the last step in cardiac physical examination, but it is by no means the least in importance. The information obtained by inspection, palpation and percussion is differentiated, extended and more definitely authenticated by auscultation.

The object of auscultation is to determine the character of the heart sounds as heard at the various valves, the cardiac rhythm, and the presence or absence of adventitious sounds.

Technic: As in auscultation of the lungs, two methods are practiced, *viz.*, mediate and immediate.

Immediate Auscultation: The immediate method is seldom used; the only excuse one has for employing the unaided ear in auscultating the heart is the unavoidable lack of a stethoscope, or to verify a faint aortic diastolic murmur. It would seem an almost impossible task properly to ascultate the apex beat of a very fat female adult.



Fig 23—Combined method of palpating and auscultating the apex beat.

Mediate Auscultation: The stethoscope should generally be employed for the examination of the heart, as with its aid the various valve areas can be definitely located, and the area of transmission is more easily followed.

Combined method of palpating and auscultating the apex beat: The systole of the heart is felt by the hand; the stethoscope conducts the apical sound through the hand.

From the sinoauricular node (S-A node) the impulse spreads wavelike over the walls of the auricles (causing them to contract simultaneously) to another specialized node or bundle of fibers located near the orifice of the coronary sinus in the annular fibers of the septal wall of the right auricle. This node is known as the

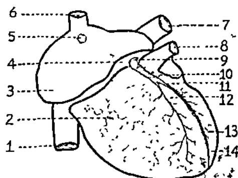


Fig 29—The conduction system of the heart. Showing the approximate relation of the more recently discovered structures to familiar anatomical divisions of the heart. 1, Inferior vena cava; 2, right ventricle, 3, right auricle, 4, atrioventricular junctional tissue, 5, sinoauricular node ("Pacemaker"), 6, superior vena cava; 7, aorta; 8, pulmonary artery, 9, node of Tawara; 10, bundle of His; 11, left branch of bundle, 12, right branch of bundle; 13, fibers of Purkinje; 14, left ventricle.

node of Tawara or the *auriculoventricular node* or *atrioventricular node* (A-V node). From the node of Tawara the impulse traverses another bundle of specialized tissue, the *auriculoventricular bundle* known as the *bundle of His*, which is the bridge that conducts the impulse from the auricles to the ventricles, causing ventricular contraction The bundle of His begins at the A-V node; it passes forward in the interauricular septum, then turns downward, and at the upper margin of the interventricular septum divides into two branches, a *left branch* that passes into the left ventricle and a *right branch* that passes into the right ventricle. Each of these branches subdivides into a network (arborisation) of fine fibers, the *Purkinje fibers*, which are distributed over the walls and papillary muscles of their respective ventricles.

While the cardiac impulse normally arises in the sinoauricular node, under certain conditions, usually pathological, impulses may arise in any part of the heart muscle. When that occurs, the normal rhythm of the heart is disturbed and various cardiac irregularities or arrhythmias occur.

The vagus nerve retards the heart rate and the sympathetics accelerate it, but neither the vagus nor the sympathetics seem to have the power to initiate or to conduct the contraction wave. The heart with its nerve connections severed may continue to beat.

When the chest is auscultated at a point corresponding to the body of the heart, two sounds are generally heard, one closely following the other, simulating a "*lubb-tup*" sound After an intermission of a fraction of a second, the two sounds are repeated That heard immediately after the longer pause is the first sound in the cycle, and is known as the *first sound of the heart* or *systole;* it corresponds to the contraction of the ventricles, the carotid impulse, the radial pulse and the apex beat. The sound following the first is termed the *second sound of the heart or diastole;* it corresponds to the contraction of the auricles or dilatation of the ventricles These two sounds are produced by different parts of the heart, and differ from each other in *quality, intensity, pitch* and *duration* They are also heard with varying intensity at different valves, the first sound being loudest at the apical area, and the second loudest at the base. The first

an accentuation of the normal sound, should be noted. Should an adventitious sound be heard here, its time and character should be investigated, and the stethoscope placed over the veins of the neck to determine the transmission of the adventitious sound



Fig 27—Auscultating the aortic valve

3. *Aortic Area:* The third area to be investigated is the second intercostal space to the right of the sternum. The strength of the sound there heard should be carefully studied, noting especially whether it equals in strength the one heard at the left second intercostal space, or whether it is weaker or stronger. Any adventitious sound heard at this orifice should be studied as to quality and time, and then followed either over the carotid arteries (when the murmur is systolic in time) or down along the sternum, gradually approaching the apex (when the murmur is diastolic in time).

4. *Tricuspid Area:* The fourth area to be auscultated is the lower part of the sternum near its junction with the ensiform cartilage If an adventitious sound is heard at this orifice, it should be followed toward the liver. As pointed out previously, the clinical areas for listening to the valve sounds do not correspond to the anatomic positions of the heart valves, because the sounds produced at the various points are carried along the course of the blood stream, and are best heard at the different areas above indicated, their audibility being due to the acute change in the course of the blood stream which occurs at these points.



Fig 28—Auscultating the tricuspid valve

The Normal Heart Sounds

Origin of the Cardiac Impulse: The normal impulse which originates the orderly contractions of the heart arises in a specialized or sensitized bundle of muscle fibers situated at the junction of the superior vena cava and right auricle beneath the epicardium. This node or bundle of muscle fibers contains nerve fibers and ganglion cells, which are connected with the vagus and sympathetic nerves, and is known as the *sinoauricular* or *sinoatrial node*. It is the "pacemaker" for the heart's contractions, which, under normal conditions, determines the rate and rhythm of the heart.

sudden tension of both semilunar valves (aortic and pulmonic), it occurs at the very beginning of ventricular diastole, therefore following the first sound after a short pause. The only factor, therefore,

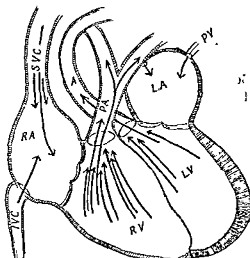


Fig 31—Systole

concerned in the production of this sound is "valvular," and is recognized by its:

Quality	Snappy
Intensity	Not very loud
Pitch	High
Duration	Short

The second sound may be represented by the syllable *"tup."* The closure of *both* the *aortic* and *pulmonic* valves produces only one sound and is heard at the apex following the first sound; this is known as the second heart sound, but each factor of the second sound may be auscultated individually when it is desired to determine the condition of either valve (aortic or pulmonic).

By listening to the aortic area (second interspace to the right of the sternum) that portion of the second sound which is produced by the closure of the aortic semilunar valves can be heard; this is known as the *aortic second sound.*

If the "pulmonic sound" is to be investigated, the pulmonic area (second interspace to the left of sternum) should be listened to; that part of the second sound which is produced by the closure of the pulmonic semilunar valve will be heard over that area.

It should be thoroughly understood that in the heart's cycle there is but *one first sound* (that caused by the closure of the mitral and tricuspid valves plus muscle sound and the impact of the heart against the chest wall) and only *one second sound,* that caused by the closure of the pulmonic and aortic valves When reference is made to the aortic second sound or to the pulmonic second sound, it is not meant to infer that there is a first pulmonic or a first aortic sound

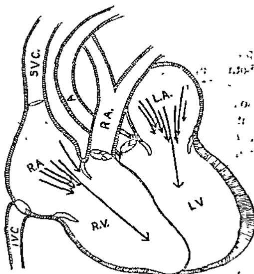


Fig 32—Diastole Blood flows simultaneously from both auricles into their respective ventricles.

Such reference can be thus explained By pulmonic second sound is meant that part of the second sound of the heart which is caused by the closure of the pulmonic valve. Aortic second sound refers to that part of the second sound

sound of the heart, or the *apical sound*, can also be heard at the base, but it is not as intense as at the apex. A *third heart sound* is occasionally heard in mid-diastole in thin chested young adults and children. It is short and very faint.

with the apical impulse (because of the heart's impact against the chest wall at that point); (*b*) it represents the systole of the heart as it occurs during the first part of the heart cycle due to ventricular contraction and auriculoventricular valve

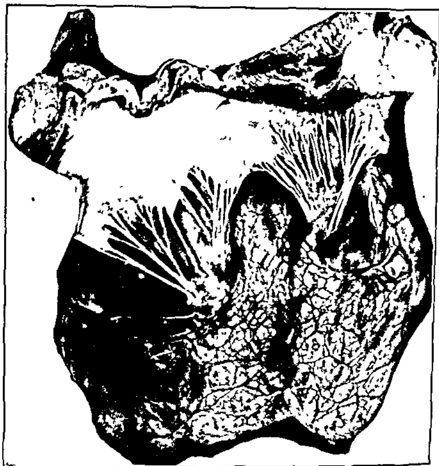


Fig. 30—Conductive system, left ventricle. (Courtesy Dr Eiman, Philadelphia General Hospital)

closure, and (*c*) the following attributes are noticeable

Quality	Loud
Pitch	Low
Intensity	Booming
Duration	Long

First Sound (Systole): This is produced by three factors: (1) The contraction of the right and left ventricles (muscular sound); (2) the closure and sudden stretching of the mitral and tricuspid valves, and (3) to a lesser extent, the impact of the heart against the chest wall.

The characteristics of the first sound are: (*a*) It is best heard over, and occurs

The first sound may be represented by the syllable *"lubb"*

Second Sound (Diastole): This is caused by the simultaneous closure and

ond sound is found in conditions which produce a loss of elasticity of the semilunar valves, as is met with in degeneration of the aortic and pulmonary orifices, or in overstretching of the aortic and pulmonary orifices, thereby preventing the valve leaflets from meeting and resisting the blood current. This is often found in aortic stenosis and regurgitation, or in pulmonic stenosis and regurgitation. Aortic stenosis will cause a very much subdued second sound at the aortic orifice, and pulmonary stenosis will have the same effect upon the pulmonic area, because of diminished tension.

Metallic quality of the second sound is heard in cases which produce accentuation of that sound, such as chronic aortitis, and also when a pulmonary cavity (under tension) is situated near the heart, or pneumopericardium; in the presence of a left-sided pneumothorax, and at times an inflated lung will help to transmit the second sound more clearly and thus add to its metallic quality.

II. Intensity — III. Pitch: One or both sounds may be *increased* or *diminished* in intensity.

Increased Intensity of Both Sounds: *Both sounds* will be *louder* in:

(*a*) *Cardiac hypertrophy,* because the heart muscle is stronger and the cavities of the heart are larger; they accommodate a greater amount of blood at each heart's cycle; the increased strength of the heart muscle causes a greater muscular sound, and the increased quantity of blood in the chambers produces more tension upon the valves, with a consequent accentuation of the valvular element of the heart sounds. Having an intensified muscular and valvular sound, therefore, a very loud first and second sound are heard.

(*b*) *Exophthalmic goiter* because of increased thyrotoxin in the blood, before the occurrence of myocarditis, cardiac action is stronger.

(*c*) *Certain anemias* in which because of the poor quality of the blood a greater quantity is required to satisfy the needs of the body; therefore, the heart has to work harder to meet the deficiency.

(*d*) *Excitement* (nervous stimulation) because of stimulation of the sympathetic nervous system.

(*e*) *Fevers*—because of toxins and stimulation of the heat-producing center, the heart often works faster and with greater force

(*f*) *Stimulation by certain drugs, e. g.*, alcohol, tea, coffee, etc.

(*g*) *Toxemiae,* though no hypertrophy be present, the louder heart sounds are caused by the rapid rate.

(*h*) *Consolidation of the lungs,* because the heart has to work against increased resistance, and also because of the presence of toxins in the blood.

(*i*) *When the lung adjacent to the heart is retracted,* an apparent increase in the loudness of the heart will be noted; the buffer being removed, the heart sounds are transmitted more readily; therefore, they sound louder than normal

Diminished Intensity of Both Sounds: Aside from extraneous causes, such as thickened chest wall, pericardial effusion and emphysematous lung covering the heart, the weakening of the heart sounds takes place in all weak heart conditions. Diminished intensity may, therefore, be found:

(*a*) In *poisoning* from various drugs.

(*b*) In *gas asphyxiation.*

(*c*) After *overexertion.*

Split Heart Sounds: The first or second heart sound may be split. Splitting of the first sound is more frequent, and is consistent with perfect health. When present, a split first sound is best heard at the height of inspiration and the *beginning of expiration, the heart sound* being somewhat prolonged and the first part of it roughened. This is followed by a momentary loss of quality and a sudden recovery; the sounds resemble the syllables *lur-eb-tup, lur-eb-tup*

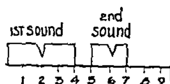


Fig. 35—Split first and second heart sounds.

A split second sound is rarely encountered, when present, it is best heard at the end of expiration and the beginning of inspiration. The split second sound often heard at the apex in mitral stenosis *is attributed by some clinicians to* vibrations produced by a rigidly constricted mitral valve, as the blood passes over it on entering the left ventricle.

A prolongation of the first sound is *often* noted in young people who present a slow heartbeat; the climax or end of that sound is sometimes considered as a third heartbeat, but in reality, it represents the final effort of the strong and slowly-contracting left ventricle to rid itself of the remaining blood in its chambers. This spurt is often visible and palpable as a distant impulse which is intensified during expiration.

Pathologic Heart Sounds

Pathologically, the heart sounds, one or both, may be altered in: (I) Quality; (II) intensity or loudness; (III) pitch; (IV) duration, and (V) rhythm.

I. **Quality:** As has been mentioned, the quality of the first sound is distinctly booming, while that of the second sound is snappy.

An increased booming quality to the first sound indicates greater strength of the muscular element, and is found in all cases of ventricular hypertrophy, because the muscle, being bigger and stronger, causes a predominance of its characteristic sound.

A *high-pitched snappy sound at the apex displacing the booming quality* means that the first sound is approaching the quality of the second. The second sound is possessed of its peculiar quality because it is a purely valvular sound; therefore, if the first sound assumes that quality, it indicates that only the valvular element of the first sound is being heard; the muscular quality being either in abeyance or entirely wanting. A high-pitched snappy first sound, which resembles the second when heard in a *rapidly beating heart, is termed embryocardia.*

A high-pitched snappy sound when heard at the apex is indicative of myocardial degeneration (fatty or fibroid); dilatation of the ventricles, and may also be heard during the course of exhaustive fevers. The reason for a high-pitched snappy sound in these *conditions* is that the heart muscle is too weak or too much thinned-out to contribute its proper element to the first sound.

The *second sound,* instead of being snappy and high-pitched, may have a *"flopping"* quality, and be rather low pitched. Since it is known that the snappiness of the second sound is caused by a certain state of tension of the semilunar valves, the fact that this snap is wanting means that the tension of the valves has diminished. A *flopping sec-*

creased intensity of the second sound is due either to hypertrophy of one or both auricles, or to increased intraäuricular tension.

Accentuation of the Second Pulmonic Sound: Accentuation of the second pulmonic sound can be recognized by its peculiar quality, which is characteristically loud, high pitched and abrupt This is heard in cases of mitral regurgitation and stenosis and in conditions which result in congestion of the lungs, such as hypertrophy of the right ventricle and pulmonary tuberculosis, pulmonary emphysema, pleural effusion, bronchopneumonia or lobar pneumonia.

Any condition that will produce increased intrapulmonary tension will cause an accentuated second pulmonic sound, because the blood in the lungs, being under greater pressure than is normally the case, the pulmonic valves snap and shut quickly with greater force and under greater tension in order to prevent a reflux, and this results in accentuation of the second sound. Mitral regurgitation and stenosis produce accentuation of the second pulmonic sound because the defect in the mitral valve gives rise to greater intrapulmonary tension, with consequent right ventricular hypertrophy.

Accentuation of the Second Aortic Sound: This condition is found in cases of increased systemic pressure and appears in disease of the peripheral circulation, hypertrophy of the left ventricle; disease of the kidneys or liver; arteriosclerosis; an atheromatous condition of, or near the aortic valve; or aneurysm of the aorta Disease of the peripheral circulation will bring about accentuation of the second aortic sound, because the blood in the aorta, being under greater pressure, causes increased resistance to the closure of the aortic valves. In order to prevent reflux of blood, the aortic valves close with a snap, as do the pulmonic valves under similar conditions. The sudden quick closure, added to the greater tension of the valve leaflets, produces this accentuation.

At times, when listening over the base of the heart, but one sound can be heard. The examiner should be painstakingly accurate in locating this sound, as often, an accentuated second sound with a weak first sound when heard at the base, will give an auditory impression of only one sound occurring at long intervals, and unless the examiner is careful, this second sound may be mistaken for the first

Weakening of the Second Sound: If increased intraäuricular pressure produces *accentuation* of the second sound, it follows that decreased intraauricular tension must produce *weakening* of the same sound. Weakening of the second sound at the base is a rather rare condition, as the intrapulmonary pressure is seldom below normal, so that any disease of the lung has a tendency to raise, rather than to lower, the pressure within the lesser circulation.

Weakening of the Pulmonic Second Sound: After a previous accentuation, this is a danger signal indicating weakness and dilatation of the right auricle. Pulmonary stenosis and regurgitation, and at times tricuspid regurgitation, when associated with right ventricular weakness, will cause a feeble pulmonic second sound A weakening of the second pulmonic sound during lobar pneumonia offers a grave prognosis, calling for active cardiac stimulation.

Weakened Second Aortic Sound: This results from decreased pressure in the systemic circulation; it may occur in

(*d*) After *hemorrhage.*

(*e*) In *acute dilatation* of the heart.

(*f*) *Before death* — in a previously good heart.

(*g*) In some *febrile diseases.*

(*h*) When *degeneration of the heart muscle* exists.

(*i*) In *coronary thrombosis.*

(*j*) In certain *nervous diseases*

Increased Intensity of the First Sound: Conditions that produce increased intensity of both sounds are largely responsible for accentuation of the first heart sound. There are two *varieties of accentuation of the first* sound:

1. When the systolic sound is very loud and booming in character, of long duration and low pitched, it indicates that the muscular quality is predominating over the valvular (found in all cases of cardiac hypertrophy).

2. The second variety presents a short, snappy, sharp sound of a higher pitch This usually occurs in a heart that has previously been hypertrophied, but is undergoing dilatation, the valvular sound predominating over the muscular quality

High pitched, short, snappy heart sounds are frequently seen in students, soldiers, and others who, after a short period of strenuous physical exertion, have settled down to a quiet and generally inactive life. Various cardiac neuroses, such as neurocirculatory asthenia, present the same quality and pitch, as does also the so-called "tobacco heart." If the accentuation is heard only over the tricuspid area, the mitral area being unaffected, it indicates right ventricular hypertrophy. Hypertrophy of this chamber very rarely presents the dull booming sound heard in left ventricular hypertrophy, chiefly because the right ventricle has a weaker muscle wall, so that the accentuation is *usually of a* "flopping" character, and, as a rule, lasts but a short time before the weakening of the muscle of the right ventricle is followed by dilatation with the consequent murmur. It is found in all cases of mitral stenosis and other conditions that increase the intrapulmonary pressure (*i. e*, emphysema, etc.).

Diminished Intensity of the First Sound: This occurs as a result of myocardial weakness. The ventricular walls, not being strong enough to contract properly and with sufficient force, produce a *sound that is weak, feeble, and lacking* in individuality. An enfeebled first sound is heard in cases of myocarditis, fatty degeneration of the heart, dilatation, atrophy and during the course of wasting fevers.

A strong booming first sound that has suddenly become "floppy" in character, is the first sign of oncoming ventricular dilatation or degeneration

Apparent weakness of the first sound is found in cases of emphysema, pleural effusion, pericardial effusion and generalized thick chest wall In these conditions the heart muscle is unaffected, but the sound is prevented from being properly heard by the interposition of *fluid or thickened tissue*

Accentuation of the Diastolic Sound (second sound): The diastolic or second heart sound is heard at its best at the base of the heart. If the second sound is louder at the apex than the first sound, it indicates ventricular weakness and auricular hypertrophy, although at times—even without existing auricular hypertrophy—the second sound may be stronger than the first This is particularly true when the ventricles are so weak that the normal auricular sound seems strong in comparison. In-

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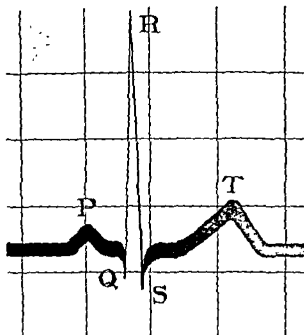
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detectable at the wrist, carotid artery or any superficial artery.

Electrocardiographic Interpretation of Heart Action

The study of cardiac diseases, particularly those affecting the heart muscle, has received great impetus from the aid of the electrocardiograph. This instrument has been the means of simplifying and explaining many pathologic conditions of the heart muscle, either hitherto wholly unknown or not fully understood. The arrhythmias, in particular, have been extensively investigated and properly classified according to their origin and mode of production For technic of electrocardiography, SEE: p 1046.

On the electrocardiogram the first sound of the heart or the systolic sound corresponds to the combined deflections of the R and T or the Q-R-S-T complex. The thrust accompanying the second heart sound or the diastolic sound is not felt in any of the arteries but may be felt in the jugular veins. At the



wrist and over the carotids it is marked as a negative period. The diastolic sound corresponds to the P wave on the electrocardiogram. The long pause between each cycle is lengthened when the heart is slow and is shortened when the heart rate is fast. In the electrocardiogram this long pause is represented by the waveless space between the T wave and the P wave of the next

general vasomotor relaxation and after severe hemorrhage or serious diarrhea.

In aortic stenosis, and often, in aortic regurgitation, resistance to the systemic circulation is, to a great extent, wanting, because of the crippled condition of the valves. The result of diminished valvular resistance is a feeble second sound, or an entire absence of that sound In mitral regurgitation and stenosis the aortic second sound is sometimes weakened, on account of insufficient tension in the aorta. Pulmonary regurgitation and stenosis may also be productive of an enfeebled second aortic sound

IV. Duration: *In a heart acting normally* the two sounds and the long pause follow each other in three-quarter or triple time, *i. e*,

First sound (one) Second sound (two)
Long pause (three)
First sound (one) Second sound (two)
Long pause (three)

However rapidly a normal heart may act, this rhythm is preserved; in disease there may be an alteration in the relative length of the heart sounds or the pause The following variations are noted:

Embryocardia: This is so called because it resembles the fetal heart sounds. The first and second pauses are of equal length, the sound resembling the regular rapid tick of a short pendulum (*tick-tock*). A second variety is an undue prolongation of the first sound, followed by an alarmingly long pause This may occur either as a result of digitalis poisoning (long diastole), or as the effort of an overworked heart, too weak to continue its labor, seen in severe myocarditis, or heart block.

Reduplication: Practically speaking, the first and second sounds of the heart are made up of two firsts and two seconds (two semilunar and two auriculo-ventricular), but they are blended by the synchronous closure of the left and right hearts. If, for any reason, the valves are prevented from closing simultaneously, we may hear three or even four sounds instead of but two sounds Such a condition may be due to faulty innervation or degeneration of that part of the heart which transmits the impulse; this is quite common in myocardial degeneration and in chronic interstitial nephritis, as well as in mitral stenosis after failure of compensation.

Rhythm

The normal cardiac rhythm is initiated at the sinoauricular node whence it passes along the sinus, sweeps over the ventricular walls to the A-V node, then traverses the bundle of His, that is, the A-V bundle, and follows its two main divisions into the right and left ventricles This procedure occurs at regular intervals and at a definite rate per minute

Auscultation of the heart at the apex beat reveals a systolic sound followed by a short pause, which is followed by the diastolic sound; this constitutes a single heart cycle. Then follows a longer pause after which the heart cycle is again heard. In the normal, such cycles occur uninterruptedly at a definite rate per minute, with certain slight variations under various circumstances The heart rate in adult males, in the sitting position, is between 70 and 76—usually about 72—per minute; it is faster when standing, and after physical and mental exertion and often after a full meal The heart rate is slower in the recumbent position, when thoroughly at rest, during sleep and in the aged In women, the heart rate is somewhat faster than in men, and it is still faster in children Each systolic heart sound is accompanied by an apical thrust and a pulse wave

detectable at the wrist, carotid artery or any superficial artery.

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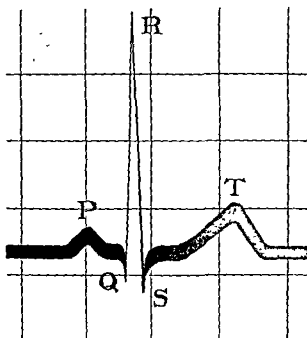


Fig 36—The primary electrocardiographic wave (schematic drawing) Normally the first evidence of heart activity arises in the auricle, and it causes a small, rounded elevation in the record which is known as the auricular wave, designated by the letter *P* Shortly afterward the ventricles [illegible]
arise a series of [illegible]
After a period [illegible]
wave *P* again [illegible]
rhythmically recurs in the actual heart record.

cycle. The short pause or the intercyclic pause is represented by the short space between the P wave and the left limb of the R wave. In the electrocardiogram the impulses as well as their rate of conduction are indicated by distinct waves which occupy a definite time in their passage from one part of the heart to the other.

The P wave represents the spread of the wave over the auricles. The summit of the P wave occurs when the impulse has reached the A-V node The interval between the beginning of the P wave to the base of the right limb (Q) of the R wave (P-R interval) represents the time consumed by the impulse in traveling from the auricles to the ventricles Normally this interval occupies no more than two-tenths of a second. The R and T waves represent the ventricular contraction.

The R wave (ventricular wave) appears as a tall spikelike prominent curve in the electrocardiogram, and should be directed upward in the first three leads and downwards in the fourth lead. Its greatest amplitude is usually attained in lead II, being from 10 to 20 millimeters. The R wave is extremely short, measuring from 0 03 of a second to 0.1 of a second. The foot of the R wave, beginning at Q and ending at S, is what is known as the Q-R-S interval.

The distance from the base of the right R line (Q) to the base of the left R line (S) is about one-tenth of a second, and from the Q line to the end of the T wave, the ventricular impulse, is about 43 hundredths of a second. The duration of the T wave in the young is about 27 hundredths of a second; in the old the T wave may be flattened out. In disease of the heart, various changes occur both in the appearance of the waves and their rate of conduction

Pathologic Variations of the Waves: ***The P Wave:*** The P wave is prominent in mitral stenosis and auricular hypertrophy. It is often bifurcated in mitral stenosis because of the disproportionate size of the two auricles.

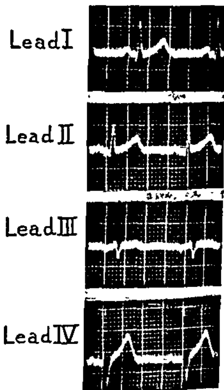


Fig 37 — The four leads Generally in Lead III the R wave is directed upwards In this Lead III the R wave is directed downward. The T wave is biphasic (not altogether normal).

It is prolonged when the excitation wave is interfered with in its passage by a hypertrophied or damaged muscle. The P wave is absent and is replaced by a number of fine oscillations in auricular fibrillation; it is distended or deflected downwards when the impulse arises in an abnormal focus and travels an abnormal course. Lengthening of the P-R interval indicates delayed conductivity

through the bundle of His (A-V bundle). Shortening of the P-R interval may be due to the impulse's arising in the A-V node instead of the S-A node

The R Wave: The R wave points upwards in lead I and downward in lead III, in left axis deviation (left ven-

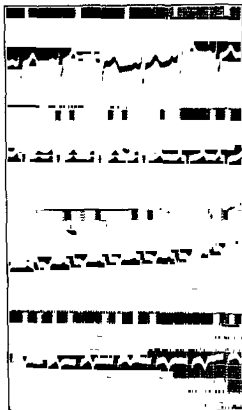


Fig 38—Right Axis Deviation (Right ventricular preponderance). (Courtesy Dr. H K. Mohler.)

tricular preponderance). It points downwards in lead I and upwards in lead III, in right axis deviation (right ventricular preponderance). In myocardial damage the R wave may be shortened or lengthened, splintered or notched, M'd or W'd.

The Q-R-S Complex: Widening of the base of the R wave (Q-R-S complex) indicates heart block either of the right or left bundle branches. In complete heart block all waves are delayed. Notching of the R wave is found in myocardial damage and in coronary disease.

The T Wave: This is inverted or flattened in severe myocarditis, in digitalis poisoning, anorexia and other toxic states. The T wave is more prominent in the young and vigorous, particularly during or soon after muscular exertion. It is flattened in the old, and often in arteriosclerotics. An inverted T wave in lead III may be consistent with good health. Its significance in an otherwise normal person is not known.

Arrhythmia*
(Disturbance of the Heartbeat)

Disturbance in the rhythm of the heart *is manifested by heart rates that are* either slower or faster than normal, or by alteration of the sequence of "systolic sound, short pause, diastolic sound and long pause" The heart sounds thus fail to follow a normal cycle and assume various abnormal patterns or irregularities. Many of these irregularities can be diagnosed by physical signs and nearly all of them show their peculiarities on the electrocardiogram

Disturbance in the rhythm of the heart or arrhythmia may be caused by various organic diseases and functional disorders which either damage the heart muscle so that it cannot conduct or respond to the normal impulses, or the impulses which initiate the heart's contractions fail to arise at their normal location or fail to traverse their normal route Disturbance in the rate and often in the rhythm of the heart may also be caused by vagus and sympathetic influence.

* For more complete discussion of arrythmias see page 510.

The arrhythmias for convenience are here divided into three groups.

I. Those associated with rapid heart action (Tachycardia).

II. Those associated with slow heart action (Bradycardia).



Fig 39—Left Axis Deviation (Left ventricular preponderance). (Courtesy Dr H. K. Mohler)

III. Those in which there is an irregularity of rate, rhythm and volume.

I. Tachycardia (rapid heart action): Rapid though regular heart action is of three varieties: (1) Sinus tachycardia; (2) paroxysmal tachycardia, and (3) auricular flutter.

1. *Sinus Tachycardia:* This consists of rapid though regular heart action, ranging from 80 to 140 or more per minute. The rate is increased by psychic disturbance or physical exertion, and may be reduced by physical and mental rest. This condition is as a rule due to the effect on the sinoauricular node by either vagus depression or sympathetic stimulation. This type of tachycardia is seen in: (*a*) Physiologic reaction to excitement, anxiety, exertion, pain, hemorrhage, shock and fevers. (*b*) Reaction to food and drugs as alcohol, tea, coffee, tobacco, epinephrine, strychnine, atropine, thyroid and other drugs that either stimulate the sympathetic or paralyze the vagus. (*c*) Thyrotoxicosis, where the pulse rate becomes easily accelerated, but does not return to normal on rest or during sleep. (*d*) Neurocirculatory asthenia, in which condition acceleration of the heart rate is more instantaneous and requires less provocation than in normal individuals though the provocative agents are the same in both. (*e*) Reaction to toxins in certain of the infectious diseases and fevers, myocarditis and certain types of heart failure.

2. *Paroxysmal Tachycardia:* This is characterized by the sudden onset of paroxysms of rapid heart action of regular rhythm. The rate may vary from 120 to 320 per minute and the paroxysm may last from a few minutes to several hours; rarely several days.

Etiology: It may be due to abnormal irritability of the heart and is brought about by various exciting factors such as fatigue, tobacco, alcohol, digitalis poisoning, sudden exertion, indigestion and anxiety. There are three types of paroxysmal tachycardia:

(*a*) *Auricular:* This is the commonest and the least important; it occurs in otherwise normal hearts

(*b*) *Ventricular:* This type is usually serious; it occurs infrequently and is

associated with heart damage and may cause pulsus alternans.

(*c*) *Auriculoventricular Nodal:* This type is very rare and comparatively benign.

The various types of paroxysmal tachycardia may be recognized by electrocardiographic study. The heart rate is not influenced by rest or by exertion.

3. ***Auricular Flutter:*** This consists of a rapid, regular rhythm or a regularly occurring irregularity. The auricular rate may be only as fast as the ventricular rate, but is usually 2 or 3 times as fast This causes a 1:1, 1:2, or 1·3 block. This condition is usually associated with heart damage The rapid impulses in their circus movement along the auricular walls are not transmitted at the same rate to the ventricles. An accurate diagnosis is made by electrocardiographic study. The heart rate may vary from 100 to 200 per minute and is not influenced by rest or by exertion.

II. **Bradycardia** (Slow though regular heart rate): This is of three types, (1) Sinoauricular; (2) auriculoventricular nodal rhythm, and (3) auriculoventricular block.

1. ***Sinuauricular Bradycardia:*** This is due to vagus influence on the sinoauricular node. The rate varies from 30 to 60 per minute. The rhythm is usually regular, though sinus arrhythmia is occasionally associated with it. This condition is not serious. It occurs as follows: (*a*) Normally in some individuals and in the aged; (*b*) during sleep or rest; (*c*) it may be induced by carotid or eyeball pressure; (*d*) by fright; (*e*) extreme cold; (*f*) as the result of intracranial pressure; (*g*) accompanying certain diseases as jaundice, myxedema, mumps, typhoid fever and at times during convalescence from influenza and during the puerperium; and (*h*) as a reaction to certain drugs, such as opium, digitalis, and physostigmine.

2. ***Auriculoventricular Nodal Rhythm:*** This is rather rare; the A-V node controls both the auricles and the ventricles. When no stimuli passes from the sinus node to the auricle (sinus block), the heart rate is generally slow, about 40 per minute; this may be induced by carotid or eyeball pressure. If the A-V node is irritated so that its impulses are propagated faster than those of the sinus node, the heart rate is fast. This may be temporarily induced by large doses of atropine.

3. ***Auriculoventricular Block:*** In complete block the auricular impulses do not traverse the bundle of His, therefore the ventricles originate their own rhythm The pulse rate may vary from 20 to 40 per minute In partial heart block the pulse rate varies The block may occur in the A-V bundle or in bundle branches Heart block is usually an indication of a diseased myocardium (SEE: p. 515)

III. **The Irregularities as to Rate, Rhythm and Volume:** These irregularities may occur with a rapid or a slow heart action They include: (1) Sinus arrhythmia; (2) extrasystoles (premature beat); (3) auricular fibrillation; (4) auricular flutter; (5) auriculoventricular block, and (6) pulsus alternans

1. ***Sinus Arrhythmia:*** This is a functional condition found in the young and is of little pathologic significance, though occasionally it may be associated with heart damage. The rate is rapid during inspiration and slows during expiration.

2 ***Extrasystoles (premature beat, premature contraction):*** The stimulus arises outside the sinoauricular node The irregularities may occur at regular intervals; they may be many or few.

The arrhythmias for convenience are here divided into three groups.

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II. Those associated with slow heart action (Bradycardia).

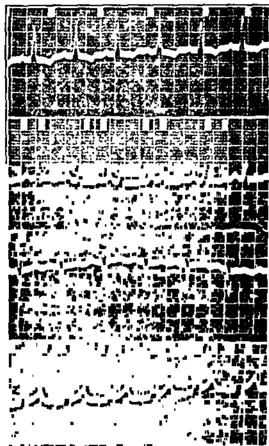


Fig 39—Left Axis Deviation (Left ventricular preponderance). (Courtesy Dr H K. Mohler)

III. Those in which there is an irregularity of rate, rhythm and volume.

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Etiology: It may be due to abnormal irritability of the heart and is brought about by various exciting factors such as fatigue, tobacco, alcohol, digitalis poisoning, sudden exertion, indigestion and anxiety. There are three types of paroxysmal tachycardia:

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(*b*) *Ventricular:* This type is usually serious; it occurs infrequently and is

CONDENSED CHART OF HEART IRREGULARITIES

Irregularity	Order of Frequency	Clinical Recognition	Significance	Treatment
Sinus Arrhythmia	Childhood Excessive tobacco Functional nervous disturbances	Rate increases on inspiration and decreases on expiration No alteration in rate when breath is held	Not pathologic Physiologic in childhood	None during childhood Sedation for nervous disturbances.
Premature Contractions	Advancing years Acute infections Digitalis "coupling" Toxemia	Occur usually when patient is at rest Heart contracts in advance of the anticipated interval, then follows a compensatory pause—prolonged diastole Irregularity greatly lessens or disappears when heart rate is accelerated by emotion or exercise	Occasional premature contractions compatible with health Progressively increasing premature contractions indicate myocardial involvement Multiple premature contractions indicate myocardial damage	Cardiac drugs not indicated Seek for, and if possible, remove systemic cause or focal infection Rest Subsequent studies to determine whether progressive in nature.
True Paroxysmal Tachycardia	Hyperthyroidism Neurocirculatory asthenia Neurasthenia	Rapid rate absolutely abrupt in onset and absolutely abrupt in termination, often without demonstrable immediate cause	Rarely die during attack. Live through successive attacks for years	Rest to conserve demands on heart muscle already inefficient as a circulatory supply, due to ventricle contracting on insufficient content of blood Seek for possible neuropathic cause Pressure on right vagus nerve in carotid sheath is effective in aborting attack perhaps in 30 per cent of cases Mecholyl is more effective.
Auricular Flutter	Myocardial fatigue Myocardial exhaustion Myocardial change	May be suspected when a rapid pulse of 180 or more is suddenly halved in rate Definite clinical recognition impossible, electrocardiographic study necessary	Acutely induced disturbance of auricular musculature	Absolute rest. Digitalis, changes auricular flutter to auricular fibrillation, after which normal rhythm ensues
Auricular Fibrillation	Acute rheumatic fever Acute infections Cardiosclerosis. Chronic infections Exopthalmic goiter.	Fibrillation Triad { 1 Absolutely irregular pulse, irregular in rate, in rhythm and in volume. 2. Disordered ventricular action 3. Pulse deficit } 4 Irregularity increases on exercise 5 Embolism, circulatory failure, "mitral facies", husky voice, visceral congestion	When acutely induced, may be fleeting and never return When corrected by drugs, marked likelihood of chronic form recurring upon slight provocation.	Absolute rest imperative until pulse deficit is reduced Digitalis brilliantly effective when given in sufficient dose. Strophanthus Dangerous if given immediately following course of digitalis Venesection for emergency of circulatory collapse
Heart Block	Digitalis, loose administration of Syphilis Diphtheria Acute infections	Strongly suggestive signs and symptoms are. 1 Pulse rate of 50 or less. 2 Sudden drop in pulse rate during course of or during convalescence from acute infections 3 Four or five waves in jugulars to one in the carotids 4 Auricular contractions which do not result in ventricular contractions may sometimes be heard when ear is laid directly on upper left chest 5 Gallop rhythm and reduplication of heart sounds (bundle-branch block). 6 Stokes-Adams syndrome 7 Rate shows but trifling increase following vigorous exercise	A. Lower grades, such as sinoauricular block or dropped beats, usually not serious but may be precursors of higher grade block B Toxic block· May be fleeting and never return, occurring during convalescence from exhausting or overwhelming illness, likely to prove promptly fatal C High grade blocks· Grave heart involvement, in which heart muscle has shared damage with conduction system Usual life expectancy, three years	A. Low grade, no treatment other than curtailment of physical activities. Perilous to employ digitalis B Toxic block Epinephrine intravenously. C. High grade· Atropine Lessen daily demands on heart muscle.
Pulsus Alternans	Myocardial exhaustion Cardiosclerosis Protracted illness.	Every other pulse wave is of less volume than its predecessor Look for a period of alternation following a premature contraction Slight pressure on brachial artery will obliterate weaker beats and thus cause sudden halving of rate at radial artery	Myocardial exhaustion Usually premonitory of the end of life.	Supportive cardiac drugs in guarded dose

Modified from "Heart Affections: Their Recognition and Treatment," by Sr. Calvin Smith. F. A. Davis Co., Publishers, Philadelphia, Pa.

These irregularities are more pronounced when the heart rate is slow. Extrasystoles may be (*a*) auricular, (*b*) ventricular, or (*c*) auriculoventricular in origin.

(*a*) *Auricular Extrasystole:* This is not very common. The premature contraction of the auricle results from an abnormal stimulus arising in the wall of the auricle before the normal stimulus arises from the sinus. The premature contraction of the auricle is usually followed by a premature contraction of the ventricle. The compensatory pause is not noticeable because, following the premature beat, a normal impulse arises in the node which causes the normal auricular ventricular contractions at normal intervals. The electrocardiogram will show a normal P-R-T sequence but of short duration, the T and P waves being quite close to each other. Occasionally auricular extrasystole may cause auriculoventricular block.

(*b*) *Ventricular Extrasystole:* This is the commonest arrhythmia Here the abnormal focus or stimulus arises in a ventricle, therefore the ventricles contract before the normal impulse from the auricles can reach them and that impulse is wasted. The ventricles do contract when the next normal auricular impulse reaches them. The interval then between the premature contraction and the next normal contraction is decidedly lengthened, causing a comparatively long pause (the compensatory pause). The premature contraction is not as strong as a normal contraction, therefore the beat following it is forcible.

(*c*) *Auriculoventricular Nodal Extrasystole:* This occurs when the stimulus arises in the auriculoventricular functional tissue and passes to both the auricle and ventricle so that they may contract simultaneously or one may precede the other. There is usually no compensatory pause unless the premature beat is an escape of the ventricle.

3. ***Auricular Fibrillation:*** This type of arrhythmia is decidedly irregular in time, volume and rate. The cardiac rate is usually fast though it may be slowed by digitalis or quinidine. The volume is variable and the force changeable. The faster the heart rate the more pronounced is the irregularity. There is usually a pulse deficit, *i e.,* the heart rate is faster than the pulse rate (For further detail and electrocardiogram, SEE: Fig 28, No. 12, pp. 512 and 520)

4. ***Auricular Flutter:*** This may follow or precede auricular fibrillation. The rate is fast and the beats may occur in regular sequence or they may be irregular. In both fibrillation and flutter the impulse circulates continuously in the auricle, more rapidly in fibrillation than in flutter. Many of the impulses fail to reach the ventricles; others are rudimentary

5. ***Auriculoventricular Block:*** In complete block the auricles and ventricles have an independent excitation area, therefore they beat independent of each other. The pulse rate may be very slow, 20 to 40 per minute, but is usually regular The auricular impulse fails to reach the ventricles; therefore they are obliged to initiate their own contractions In partial block or in branch bundle block the rhythm is often irregular.

6. ***Pulsus Alternans:*** This is a condition in which a full pulse or a strong heartbeat alternates with a weak pulse or a weak heartbeat. The rate may be rapid or slow, and the alternations are regular. This condition is found in severe myocardial weakness.

stair climbing. Normally there will be an increase of 20 beats per minute in the pulse, and the blood pressure will rise from 8 to 10 mm Hg. Insufficiency of the myocardium will increase the pulse rate to from 20 to 30 beats per minute, but the blood pressure rise will be slower, averaging about 6 mm. Hg or less. This rise may be quickly followed by a fall below normal, or, on the other hand, there may be no preliminary rise at all. The length of time required for the pulse rate and systolic pressure to return to the normal may be taken as a measure of the amount of cardiac insufficiency present.

A modification of this test is the "hopping test," in which the patient is required to hop 20 to 50 paces on one foot, comparisons of pulse rate and blood pressure being made as in the staircase test. This test is not as satisfactory as the first, because in the hopping test the amount of work done cannot be gauged with the same accuracy with which the amount of energy expended in climbing a flight of stairs, the exact height of which is known, can be measured. The amount of work done in foot pounds is equal to the weight of the individual in pounds divided by the number of feet ascended.

Patients obviously too ill to climb stairs or to hop may be given milder forms of execise, such as walking across a room a certain number of times. Those in bed should have their exercise restricted to raising their arms several times, or turning in bed, of sitting up in bed or sitting on a chair placed near the bed. The amount of exertion is to be increased according to the obvious condition of the patient.

(*b*) ***Graupner's Test:*** It was observed by Graupner that when the pulse rate has risen after exertion and again fallen to normal, the systolic pressure gradually rises to a maximum, usually reaching it in about six minutes, with a subsequent decline to normal which occupies about 18 to 20 minutes. If the heart is seriously weakened, this rise of blood pressure following the pulse rise, may be altogether absent, the pressure declining from the start, and thereafter gradually rising once more to normal In healthy individuals the pulse will reach its maximum in from five to ten minutes. To perform this test, the patient is instructed to turn a wheel which is supplied with a brake and an adjustment for measuring the amount of energy expended. This specially designed apparatus is known as the Zuntz ergometer. The tests are repeated for several successive days, always at the same time of day, noting the pulse rate, blood pressure and size of the heart both before and after each test. The patient must not be excited in any way while undergoing the test, and should not be urged to exert himself to the point of exhaustion The apparatus mentioned requires thigh muscles work, but other machines have been devised which make use only of the arm muscles Graupner's investigations led him to conclude that if the blood pressure remains constant after the exercise, the heart muscle is sufficient If the blood pressure falls after using the machine, there is some cardiac insufficiency present If the blood pressure rises but soon returns to normal, there is compensatory sufficiency, but if the blood pressure rises, and then falls without any tendency to a subsequent rise, it demonstrates fatigue of the heart muscle. It was his belief that if the pulse is accelerated and the patient becomes "short of breath"

Functional Tests for Determining Cardiac Capacity and Reserve

Much stress is laid on examination for the diagnosis of a normal heart and the various deviations from the normal, so that one may recognize cardiac enlargement, various irregularities, murmurs and other diseases of the heart. Important as these examinations are, they often fail to reveal the cardiac reserve power, that is, the amount of reserve stored up in the heart muscle which permits it to respond to prolonged or unusual strain It is important to gauge the functional capacity of the heart in those about to assume laborious occupations to which they are not accustomed, or in athletes to be chosen for specially strenuous or competitive tasks Cardiac capacity tests are most important for patients convalescing from acute ailments, from acute myocardial disease, from coronary infarctions and from other conditions that cause cardiac embarrassments In these the usual listening to the heartbeat, the mapping out of the size of the heart, the checking of the blood pressure, and even the securing of an electrocardiogram are inadequate for determining the functional or reserve capacity of the heart.

There are several groups into which tests of cardiac function may be divided. The following classification has been modified from Barton to show how the various tests are to be placed in these four categories:

I. *Reaction to Muscular Exertion, Active or Passive, as a Basis for Estimating Cardiac Function* (*a*) The staircase test; (*b*) Graupner's test; (*c*) Mendelsohn's test; (*d*) Katzenstein's test; (*e*) Hertz's self-checking test; (*f*) Gymnastic resistance test; (*g*) The holding the breath test; (*h*) The venous pressure test; (*i*) the vital capacity of the lungs.

II. Application of cardiac reflex estimation in determining heart function Merklen's test.

III. Estimation of sodium chloride elimination as a test of cardiac sufficiency. Vaquez-Digne test.

IV. Modern clinical and instrumental methods of investigating cardiovascular conditions; their applicability to estimating cardiac function.

1 The sphygmomanometer as an index of cardiac function (work velocity ratio; sphygmobolometry, sphygmobolography, energometry, etc.).

2 Roentgenoscopy and roentgenography as indices of cardiac function.

3 Sphygmocardiography and electrocardiography; their relation to cardiac functional capacity.

I. Reaction to Muscular Exertion: In this type of test one must consider chiefly the rate of the pulse, the blood pressure (systolic and diastolic) and the area of cardiac dullness or the size of the heart (percussion, roentgenography). All these methods have the common defect in that individual differences will produce quite different results by the same tests, and that such factors as the state of the nervous system, the mode of life in regard to the amount of regular physical exertion undergone, the size and general muscular state and strength, may markedly influence the results obtainable. However, if proper allowance is made for such individual factors, all the tests are of value

(*a*) ***Selig's "Staircase Test":*** The pulse and the systolic pressure are taken and the patient is to ascend a *flight of* steps rapidly. The pulse and the systolic pressure are taken again after *the*

stair climbing. Normally there will be an increase of 20 beats per minute in the pulse, and the blood pressure will rise from 8 to 10 mm. Hg. Insufficiency of the myocardium will increase the pulse rate to from 20 to 30 beats per minute, but the blood pressure rise will be slower, averaging about 6 mm. Hg or less. This rise may be quickly followed by a fall below normal, or, on the other hand, there may be no preliminary rise at all. The length of time required for the pulse rate and systolic pressure to return to the normal may be taken as a measure of the amount of cardiac insufficiency present.

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after he has done work amounting to the equivalent of from 45 to 300 kilograms, the heart is evidently insufficient. The ordinary bicycle, made stationary, will serve as a machine for testing cardiac capacity.

(*c*) ***Mendelsohn's Test:*** This test as performed by its originator, requires the use of the Gaertner ergostat, though any exercise such as stair climbing or hopping may be substituted. The pulse is carefully counted in the standing, sitting and recumbent postures, and the figures noted. This may be repeated several times so that an average may be estimated. After the performance of his given task, the patient immediately resumes the recumbent posture, and the examiner notes the time required for *the pulse to return to the normal for that posture* Mendelsohn contended that unless there is a well-marked difference between the pulse rate in the recumbent and erect position, the heart is incompetent. When resting after *strain, the competent heart returns to* normal at once. A disturbance of rate with failure to return immediately to normal following the expenditure of from 25 to 50 kilograms of work indicates cardiac insufficiency.

When a normal individual rises from the reclining to the standing position, the increase in the heart rate ought not to exceed 20 beats. Should it rise above 20 it may be assumed that the myocardium is insufficient. This is a simple test, and has considerable value, but sometimes it may be nullified by existing psychic influences, and it has also been noted that a false increase often occurs in those presenting enteroptosis.

(*d*) ***The Katzenstein Method:*** In cases of cardiac insufficiency, Katzenstein found a lowering of the blood pressure and a simultaneous increase in the pulse rate, both of which deviations from the normal appeared to maintain a proportionate relation to the incompetency of the heart muscle. The test consists of putting the patient in a reclining posture and taking the pulse rate and blood pressure. An assistant then applies pressure with his fingers for a period of from two and a half to five minutes in the groins over both femoral arteries, or—if no assistant is to be had—an Esmarch bandage may be used; after which the pulse rate and blood pressure are again recorded. If *the myocardium is sufficient the pulse* rate will be found to be diminished, and the blood pressure will rise from 5 to 15 mm. Hg. If the heart is en*larged, but still efficient, the pulse rate* will diminish or remain unchanged, and the blood pressure will increase from 15 to 40 mm. Hg. If a moderate latent cardiac insufficiency exists blood pressure and pulse will remain unchanged, *or possibly the pulse rate will increase* slightly. In greater cardiac insufficiency the pulse rate increases while the blood pressure sinks. Norris does not regard this test as of great value when used alone, but deems it useful as a corroboratory evidence *In severe cardiac weak*ness the performance of this test may occasionally be dangerous.

(*e*) ***Hertz's Self-checking Test:*** The patient is placed in a sitting posture and remains so until the pulse rate has become constant. He is *then directed* to contract the muscles of the hand and forearm with all his force, performing the *motions* slowly, paying strict attention to the performance and endeavoring to antagonize his movements as forcefully as possible. *In healthy* persons, the pulse rate is unaffected, but

if the heart is weak the rate will be increased 5 to 20 beats a minute.

(f) Gymnastic Resistance Test: This consists of noting how much exercise against resistance and for how long a time it may be performed by the patient before he shows definite signs of tiring. The rapidity of the respiration and pulse and also the blood pressure are noted.

(g) Holding the Breath Test: The length of time the patient is able to hold his breath during rest and during certain exercises is noted. In the absence of pulmonary disease this test is of some value. The more severe the cardiac damage the shorter is the time the patient can hold his breath.

(h) The Venous Pressure Test: This depends upon the occurrence of cyanosis and the degree of venous distention occurring during exertion; the weaker the myocardium the greater the cyanosis and venous distention (SEE: p. 447).

(i) Vital Capacity of the Lungs: Another fairly good test for cardiac reserve is the determination of the vital capacity of the lungs. A reduction of the vital capacity is an early sign of myocardial inadequacy.

The test is carried out as follows: The subject stands erect holding the mouthpiece of the spirameter in the mouth (care to be taken to avoid leakage). He is urged to take the deepest possible inspiration and then with the valve properly adjusted and the nose compressed he is to exhale through the mouth all the air he possibly can Five or six such deep inspirations are followed by that many deepest possible expirations. The highest reading on the scale is taken as his vital capacity. This figure is compared to standard tables for age and sex. This test is of value only in the absence of any pulmonary or bronchial disease and in the absence of fever. To be of value this test is to be repeated daily for several days and the mean vital capacity taken.

Holding one's breath while performing certain exercises, such as swimming, walking upstairs, walking across the room, or performing certain calesthenics is an adequate test for vital lung and heart capacity. Decreased exercise tolerance when the breath is held or otherwise indicates diminished cardiac capacity.

II **The Cardiac Reflex Estimation as an Index to Cardiac Capacity:** ***The Merklen Test:*** This is the best known; it makes use of Abrams reflex which consists of diminution of the area of cardiac dullness following the vigorous rubbing of the precordium; and of the Livierato reflex which is supposed to increase the area of cardiac dullness following percussion over the epigastric region If after rubbing the precordium with a roughened cloth the area of cardiac dullness does not diminish or after percussing or stroking the epigastrium the area of cardiac dullness does not increase, there is indication of myocardial damage since the reflexes do not respond in a normal way. (To attempt to judge cardiac capacity by these reflexes is of no value)

III. **Estimation of Sodium Chloride Test: Vaquez-Digne Test:** This test was based on an old premise that in myocardial insufficiency there is a lowered sodium chloride estimation. The test consists of giving a certain quantity of sodium chloride by mouth or intravenously and noting its rate and quantity of elimination. In severe myocarditis, edema may result from excessive salt intake. (This is a cumbersome test of no special value.)

IV. **Instrumentation Tests:** ***The Sphygmomanometer:*** This is an instrument devised for determining the systolic and diastolic blood pressure. The data obtained from its use is valuable (SEE: p. 413). A high systolic pressure (above 160) is a warning signal and calls for decreased exertion.

Sphygmobolometry: This was advocated by Sahli; it consists of determining the amount of oscillation of the mercury column or the needle when the blood pressure cuff is inflated to a point just above the region indicated by the diastolic pressure. It practically means *the oscillometric reading.* The instrument devised by Sahli and the methods of determining the exact pressure in the blood vessels are too complicated for clinical use.

X-ray Study: This will determine the size and shape of the heart, the comparative size of the heart to the chest wall and the sizes of the aorta, auricles and ventricles.

Electrocardiograph: This is capable of recording the heart rate and rhythm and, to some extent, the integrity of the myocardium. For electrocardiograph, polygraph, etc., SEE: p. 1045.

V. **Circulation Time (Circulation Rate):** In order to determine the velocity of the blood flow, certain substances are injected intravenously at one site and the time it takes for their detection at another site is noted. The time required for the detection of the injection substance is known as the circulation time.

The distances measured are the (1) arm to tongue time, (2) arm to lung time, (3) arm to arm time, and (4) arm to heart and pulmonary circulation time.

(1) Arm to tongue time: The patient is to assume the recumbent posture; the right or left arm is held on a level with the right auricle and one of the various solutions is injected into a vein in the antecubital fossa, and the time is noted (by stop watch) from the moment the last of the injection has entered the vein until it is detected in the back of the throat and by the tongue. The solutions commonly employed are: (*a*) Decholin[1] (4 cc. of 20 per cent solution); the normal time is 14 to 19 seconds. (*b*) Calcium gluconate[2, 3] (4 cc. of 20 per cent solution); the time from the instant the injection is begun until the sensation of heat is felt in the throat *is 8 to 16.5 seconds.* Saccharine[4] (5 cc. of a 1 per cent solution); the time from the beginning of the injection until a sweetish taste is perceived by the tip of *the tongue is 9 to 17 seconds.* Several other substances are employed for this test; each of the substances has its own circulation time. Therefore, if the test is to be of any value, the examiner should familiarize himself with the circulation time of one type of these solutions and use this one type of solution consistently.

(2) Arm to lung time: Here various volatile solutions are employed. Those in common use are ether and paraldehyde. Ether[2]: 5 m of ether is diluted with an equal part of normal saline solution and injected into the vein of an arm, as previously described. The time is calculated from the moment the injection is begun to the instant the ether is perceived in the upper respiratory passage and the individual coughs or perceives the ether. *The normal time is 3 to 9 seconds.* Paraldehyde[3]: 1.4 cc. of paraldehyde is injected in the usual way. The time the substance reaches the lungs is indicated by cough; it averages about 6 seconds.

(3) Arm to arm time: According to Koch,[4] this is obtained by injecting fluorescein into the vein of one arm and collecting at frequent intervals from a vein in the opposite arm blood samples which are examined for fluorescein. The time the first positive specimen is obtained after the injection is considered as the circulation time. Normally this fluctuates between 12 and 26 seconds, the average being 21 seconds.

(4) Arm to heart and pulmonary circulation time: According to Blumgart and Weiss,[3] this consists of injecting radium emanation into a vein and detecting its presence by a suitable apparatus at various points in the body. The time elapsed in the detection of the substance from one point to another is the circulation time for that distance.

Interpretation of the Circulation Time Tests

The circulation time is *prolonged* in heart failure, heart block, polycythemia, hypothyroidism (myxedema) and any condition that slows the circulation. The circulation time is *shortened* in paroxysmal tachycardia, auricular flutter, hyperthyroidism, and exophthalmic goiter In bronchial asthma, emphysema and mediastinal conditions not associated with heart failure, the circulation time may be *normal*.

The Venous Pressure Tests: Venous pressure may be determined by physical means and by instrumentation.

(1) By physical means the venous pressure cannot actually be measured, but sufficient information may be gathered to judge the approximate amount of stasis in the venous system. The veins usually chosen for this are the external jugular veins A normal person lying flat on his back will show distention of these veins to a level just above the clavicles When the head is raised, venous distention disappears, and when lowered below the level of the manubrium, the veins fill to a higher level. In right-sided heart failure the external jugulars are filled to a very much higher level than in the normal, both when the head is lowered or raised. The height of the column may indicate the degree of right-sided heart failure

By the instrumental or direct method, venous pressure can be measured in millimeters and is therefore a fairly accurate gauge for determining the amount of right-sided heart failure.

The apparatus consists of a glass monometer graduated in centimeters or millimeters, to which a large bored intravenous needle is attached by a rubber tube. With the patient in the recumbent posture and the arm on the level with the right auricle, the site of a large vein in the cubital fossa is sterilized and the needle is inserted into the vein The height to which the blood rises in the monometer indicates the venous pressure. To prevent clotting the apparatus may be immersed in a 2 per cent sodium citrate solution just before it is used. Several types of monometers are on the market; the principle upon which they work is the same.

The normal venous pressure varies between 6 and 10 mm, though it may be somewhat higher or lower. After exertion the pressure rises Excluding local venous obstruction the general rule is that the severer the degree of right-sided heart failure, the higher is the venous pressure.

[1] Kramer, D: Jour Physiol. Proc, 85, 1935
[2] Baer, S · Ann. Int Med, 13 · 2246, 1940
[3] Blumgart, H L, and Weiss, S.: J. Clin. Invest, 6 103, 1928-29.
[4] Koch, E.. Deut. Arch f. Klin. Med, 140, 39, 1922.

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Acquired Organic Murmurs

1. *Regurgitant murmurs* are due to the regurgitation of blood back to the chamber whence it came, because of insufficient closure of the valve leaflets.

2. *Stenotic murmurs* are due to a partial obstruction to the flow of blood at the entrance to its orifice, as a result of a stenosis of the valve orifice caused either by an inflammatory process or by vegetations upon the valve leaflets, thus preventing them from opening at the physiological moment.

(*a*) A *systolic murmur* occurs during the time of ventricular systole, that is, the time during which the ventricles contract; therefore, it is coincident with the first sound of the heart and the radial and carotid pulse.

Such a murmur may either entirely displace the first sound, or it may occur with it and continue a short time after the heart sound ceases to be heard. The following murmurs occur during systole: Stenosis of the aortic or pulmonary valves, and regurgitation of the mitral or tricuspid valves

(*b*) A *diastolic murmur* occurs at the time the auricles contract and the ventricles dilate (during the diastole); it is heard instead of or with the second sound of the heart over the valve so affected Diastolic murmurs occur as a result of a regurgitant lesion in either of the semilunar valves and also in stenosis of the mitral or tricuspid valves

(*c*) A *presystolic murmur* occurs during the last part of the diastole, when the final spasm of the auricles forces out their last remaining blood. This murmur is heard just before the first sound and ends with the systolic shock; it is caused by stenosis of the mitral valve and, *rarely, of the tricuspid valve;* at times these murmurs may be diastolic.

Systolic Murmurs

At the Apex:
1. Mitral regurgitation.
2. Due to mitral insufficiency (organic or functional).
3. Occasionally transmitted from aortic stenosis

At Aortic Orifice:
1. Aortic stenosis.
2. Aortitis, atheroma of aorta, arteriosclerosis.
3. Aneurysm of aorta

At Pulmonic Orifice:
1. Pulmonary stenosis.
2. Patent ductus arteriosus.
3. Interventricular septal opening
4. Patent foramen ovale (rare).
5. Functional murmurs
6. Often in children and young thin adults due to sudden filling and distention of the pulmonary artery.

At the Tricuspid Area
1. Tricuspid regurgitation.

Diastolic Murmurs

At the Apex:
1. *Mitral stenosis (presystolic and diastolic)*
2. Austin-Flint murmur in association with aortic regurgitation.
3. Transmitted from aortic regurgitation

At Aortic Orifice.
1. Aortic regurgitation
2. Aneurysm of aorta (continuous hum)
3. Thyrotoxicosis (rare).
4. Arterial hypertension (rare).

Pulmonic Area·
1. *Pulmonary regurgitation.*
2. Graham-Steele murmur.
3. *Transmitted from aortic area.*
4. Aortic aneurysm (to-and-fro murmur).

Tricuspid Area
Tricuspid stenosis.

Characteristics of Organic Murmurs: Since an organic murmur occurs as a result of some crippled condition of a given valve, it is important to recognize and isolate the valve or valves so *affected. This is best done by taking* into consideration the following charac-

CHAPTER XVI

Cardiac Murmurs

The various heart sounds so far considered have been modifications of the normal heart sounds due in most cases to disease of the myocardium or to the cardiac innervations, in each instance only the first and second sounds being heard, though with altered relations to each other. We shall now consider a variety of sounds occurring either before, with, or after the first or second sound or else entirely displacing them.

These *adventitious sounds,* if caused by some intracardiac condition, are termed *endocardial murmurs* or simply *murmurs* If the adventitious sound is extracardiac in origin, as for example pericardial or pleuropericardial, it is called a *friction sound.* If it is of venous or arterial origin, it is designated a *bruit* or *hum.*

Normally, the blood passes through the valve orifices without any audible sound other than those recognized as the first and second sounds of the heart, *i. e., lubb-tup* But if the normal relation of the heart valves, the composition of the blood, or the rapidity of the blood stream is altered, "eddies" will arise, which form the so-called "fluid veins," the sounds of which may be heard on the surface of the chest as murmurs

Murmurs are divided into: (*a*) Organic or valvular; (*b*) nonorganic or functional (sometimes termed hemic, anemic, dynamic and accidental)

Organic Murmurs

An organic murmur is an abnormal sound heard over the precordium because of the existence of some abnormal condition within the heart produced by an irreparable valve defect. It is the result of some *abnormal* condition of a valve which interferes with the normal circulation of the blood, either by *obstruction,* not allowing the blood to enter a chamber freely (an obstructive or stenotic murmur), or by its *inability to approximate properly,* at a time when it should be closed, and thus allowing a portion of blood to regurgitate to the cavity whence it came (*regurgitant or insufficiency murmur*).

It is obvious, if a stenotic or regurgitant murmur is caused by a lesion in a valve, that it is possible to have as many lesions as there are heart valve orifices, multiplied by two. Therefore, two lesions at each valve, namely:

Mitral Orifice
  Mitral regurgitation and mitral stenosis
Aortic Orifice
  Aortic regurgitation and aortic stenosis
Tricuspid Orifice.
  Tricuspid regurgitation and tricuspid stenosis.
Pulmonic Orifice·
  Pulmonary regurgitation and pulmonary stenosis

There may also be a double murmur in the same valve (regurgitation and stenosis), or a combination of one or two murmurs at two or three valve orifices

Classification of Organic Murmurs. Organic murmurs are classified both according to the kind of lesion producing them and according to the stage of the heart's cycle during which they occur.

Organic murmurs may be *acquired* or *congenital.*

Second: It occurs during the *systole,* at the time the left ventricle is supposed to force its blood into the aorta, and the mitral valve should be closed; since the murmur is mitral it means that the mitral valve is affected, and instead of being closed it must be open, otherwise there would be no murmur. An open valve at this time would cause a regurgitation of blood into the chamber whence it has just come, therefore, evidently the murmur is a *mitral regurgitant murmur* (SEE Fig 1).

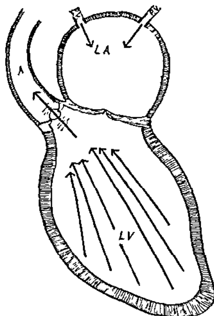


Fig 3—Aortic stenosis

Mitral Area, Mitral Murmur, Presystolic in Time: During that period of the diastole which is designated as presystole, the auricle with a spasmodic effort attempts to force its remaining blood with greater rapidity through the mitral orifice If a murmur occurs at this time, it must mean that the effort of the auricle is meeting with some obstruction, and does not allow free entrance of blood to the ventricles; consequently, the lesion must be that of *mitral stenosis* (SEE: Fig 2).

Aortic Stenosis: If a murmur is best heard over the aortic orifice (second interspace to the right of the sternum), that murmur is of necessity an aortic murmur. If this murmur occurs during the systole, it must be because of some difficulty attending the entrance of blood into the aorta, since during the systole of the left ventricle the blood enters the aorta. As the murmur occurs at this time it must be only because of some interference or obstruction to the normal flow at the aortic valve; therefore, this murmur is attributed to aortic stenosis. The aortic second sound is weak because of loss of elasticity in the aortic valve (SEE: Fig 3).

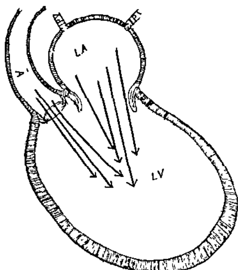


Fig 4—Aortic regurgitation.

Aortic Regurgitation: On the other hand, a murmur that is heard at the aortic orifice, or to the left of it, which is diastolic in time, must be due to a different type of lesion than that caus-

teristics: (I) Point of maximum intensity; (II) time of occurence; (III) area of transmission; (IV) quality; (V) degree of cardiac hypertrophy.

I. ***Point of Maximum Intensity:*** A murmur occurring as a result of a defective valve is heard loudest over the

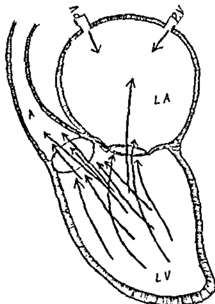


Fig. 1—Mitral regurgitation.

clinical location of that valve orifice, *i. e.*, *mitral* murmur over the apical impulse; *tricuspid* murmur over the lower portion of the sternum; *aortic* murmur in the second interspace to the right of the sternum or at midsternum, at times also in the left third intercostal space near the sternum; *pulmonic* murmur in the second left interspace close to the sternum.

When listening to the heart for murmurs, the clinical valve orifices should be systematically auscultated. If a murmur is heard with greatest intensity over the mitral area, it is evident that the mitral valve is at fault, and if the intensity of the murmur is greatest at the tricuspid, aortic or pulmonic areas, it indicates that the defect is located at one of these valves. By auscultating the valve orifices, it may be learned which of the valves is affected, but it is impossible to recognize the type of lesion. In order to determine the type of lesion, *i.e.*, stenotic or regurgitant, the second point must be considered, namely:

II. ***Time of Occurrence of the Murmur and Its Mechanism:*** As has been mentioned above, by timing is meant ascertaining whether the murmur is systolic, diastolic or presystolic. By combining the area of maximum intensity with the time of the murmur, it may be judged which valve is affected and the kind of lesion affecting it.

Mitral Regurgitation. If a murmur is best heard at the apex, and it corre-

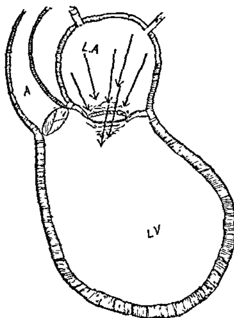


Fig. 2—Mitral stenosis.

sponds to the first sound of the heart. the systole, the following inferences may be drawn.

First: The murmur is heard at the *apex*, therefore, the *mitral valve* must be diseased (mitral murmur).

Second: It occurs during the *systole,* at the time the left ventricle is supposed to force its blood into the aorta, and the mitral valve should be closed; since the murmur is mitral it means that the mitral valve is affected, and instead of being closed it must be open, otherwise

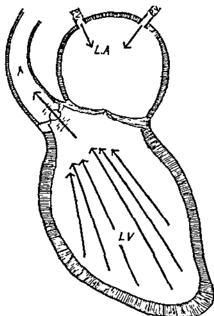


Fig 3—Aortic stenosis

there would be no murmur An open valve at this time would cause a regurgitation of blood into the chamber whence it has just come, therefore, evidently the murmur is a *mitral regurgitant murmur* (SEE Fig 1).

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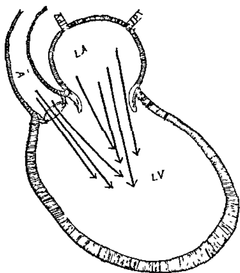


Fig. 4—Aortic regurgitation.

Aortic Regurgitation: On the other hand, a murmur that is heard at the aortic orifice, or to the left of it, which is diastolic in time, must be due to a different type of lesion than that caus-

ing the preceding one. That the aortic valve is also at fault here is beyond dispute, because the murmur is heard at the aortic orifice; it occurs during the diastole or dilatation of the left ventricle, at a time when the aortic valve should be closed while blood is

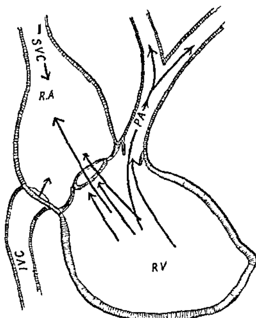


Fig. 5—Tricuspid regurgitation.

flowing into the ventricles from the auricles. If a murmur is heard at this time over the aortic orifice, it indicates that there is something wrong with the aortic valve. When the aortic valve is closed during the diastole no murmur is audible at the aortic orifice; therefore the inference is that it must be open in order to produce this sound. An open aortic valve, when the ventricle is in diastole, must necessarily cause the blood to regurgitate from the aorta into the left ventricle; hence, the murmur at the aortic orifice during the diastole is due to aortic regurgitation (SEE: Fig. 4).

Tricuspid and Pulmonic Murmurs: Murmurs heard at the *tricuspid* and *pulmonic* orifices are likewise isolated and the same reasoning holds true. It should be remembered that both auricles and both ventricles work synchronously; therefore, a stenotic or regurgitant lesion at the tricuspid orifice will have the same time as a mitral lesion; they can be differentiated because they are heard at different portions of the chest, *viz.*, the mitral murmurs over the mitral area, and the tricuspid murmurs over the tricuspid area.

With pulmonic murmurs the same holds true.

A *systolic murmur* heard at the second interspace to the left of the sternum is usually due to *pulmonary stenosis* and a *diastolic murmur* over the same area to *pulmonary regurgitation.* A pre-

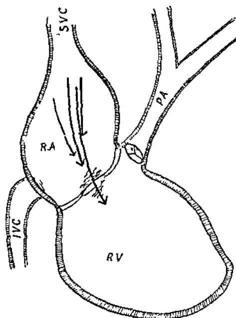


Fig. 6—Tricuspid stenosis

systolic murmur at the tricuspid area is caused by *tricuspid stenosis,* and a systolic murmur over the same area by *tricuspid regurgitation.*

Signs other than those obtained by auscultation, such as venous or arterial

engorgement, hypertrophy of the heart, the pulse, etc, must be taken into consideration when murmurs are to be differentiated (SEE · Figs 5, 6, 7, and 8).

III. ***Area of Transmission:*** In order to facilitate the recognition of murmurs and to isolate them, if several

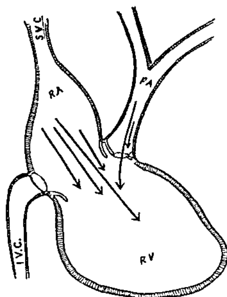


Fig 7—Pulmonary regurgitation.

are audible in the same individual, the sound must be traced from the point of greatest intensity to the point where it is entirely lost From its point of origin the sound produced by a lesion is carried with diminishing intensity along the course of the blood stream

Mitral Stenosis: This murmur is heard a little above the apical impulse, fourth interspace and a little outside of the parasternal line (near the anatomic location of the mitral valve). It is transmitted a short distance around its area, probably because the jar there produced by the stenosis is not communicated beyond the heart cavity.

Mitral regurgitant murmurs are best heard at the apex, whence they are transmitted to the left axilla and often as far back as the angle of the left scapula. This is probably because the noise is created in the left side of the heart (auricle and ventricle) and because the left side of the heart is nearest the left axilla and the left posterior aspect of the chest; at those locations the murmur may be heard, though faintly.

Aortic stenosis is best heard at the aortic orifice as a systolic murmur, and is transmitted to both carotids; it is heard louder on the right side of the neck than on the left; probably because the innominate and carotid arteries are given off from the aortic arch at an angle, so that it is easiest for the sound to travel in that direction.

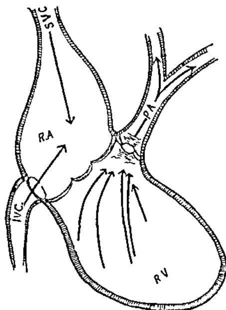


Fig 8—Pulmonary stenosis

Aortic Regurgitation: The diastolic murmur heard at the aortic orifice is transmitted downward along the sternum toward the apex, because in this lesion the blood regurgitates from the aorta

into the left ventricle and the sound goes with it, from the aortic orifice towards the apex of the heart.

Tricuspid Stenosis: A presystolic murmur is heard over the tricuspid area, but not transmitted (analogous to mitral).

Tricuspid Regurgitation: A systolic murmur is heard at the tricuspid orifice, transmitted to the right, and often audible over the liver.

Pulmonic Stenosis: A systolic murmur can be heard at the pulmonic orifice, transmitted to the veins of the neck.

Pulmonic Regurgitation: A diastolic murmur is heard at the pulmonic orifice, transmitted downward toward the right ventricle.

IV. ***The Quality:*** All stenotic murmurs are harsh and churning in quality, because such a murmur occurs as the result of an obstruction; force exerted against resistance will cause a greater amount of vibration. All regurgitant murmurs are softer and are blowing in character, for they are due to leakage and not to increased resistance as is the case with stenotic murmurs.

V. ***Degree of Cardiac Hypertrophy:*** The occurrence of a murmur is significant of some valvular defect, which of necessity must interfere with the normal quantity of blood thrown into the circulation. In order to compensate for this shortage, the heart chamber affected by the disorder increases in size and its walls hypertrophy so as to be able to accomplish more work than in its normal state

Mitral Regurgitation: The leakage in the mitral valve at first causes a diminished quantity of blood to be forced into the aorta, but at the next diastole a greater volume of blood is forced into the left ventricle, this blood being a portion of that which has been previously regurgitated added to the normal amount; the left ventricle, therefore, has a larger quantity of blood to deal with, and working as it does under disadvantages, it must of necessity hypertrophy in order to maintain the circulation (SEE: Fig. 1, p 450).

Mitral Stenosis: In this lesion an insufficient amount of blood enters the ventricle. The left auricle has to work against resistance, in order to overcome the obstruction; consequently hypertrophy of the left auricle and right ventricle is produced The left ventricle is not changed in size; though at times it may show signs of atrophy. The presence of a hypertrophied ventricle in mitral stenosis may be due to a preëxisting mitral regurgitation or to rheumatic myocarditis (SEE: Fig 2, p. 450).

Aortic Stenosis: This lesion causes the left ventricle to work against a resistance even greater than that of mitral regurgitation; therefore the left ventricular hypertrophy is greater in aortic stenosis than in mitral regurgitation (SEE: Fig. 3, p. 451).

Aortic Regurgitation: This lesion produces the greatest amount of left ventricular hypertrophy, because at each systole the left ventricle has to cope with a double quantity of blood, *i. e.* the normal amount brought to it through the mitral valve during the diastole and the quantity that regurgitates at the same time through the incompetent aortic valve. The hypertrophy is often so great that the heart in this condition is called *cor bovinum* or *ox heart* (SEE: Fig. 4, p. 451).

Pulmonic murmurs and tricuspid regurgitation will cause right ventricular hypertrophy, because the strain of the circulation falls upon that chamber in

the presence of those valvular defects But the hypertrophy of the right ventricle never reaches to the same proportion as does the left ventricle, because the right ventricle is thinner and has less compensatory power. After the hypertrophy has reached its maximum, overstrain will cause that chamber to dilate, which produces heart failure, or "ruptured compensation"

Combined Murmurs: By combined murmurs is understood the occurrence of two or more murmurs in the heart; they are recognized by noting: (I) Area of greatest intensity of each murmur, (II) the time of occurrence; (III) the respective areas of transmission; (IV) their respective qualities

Résumé of Organic Murmurs, Single and Combined

MITRAL REGURGITATION (See Fig 1)

Area of greatest intensity. At apical impulse.
Time Systolic
Transmitted: To left axilla and beyond
Quality Blowing
Accentuation of pulmonic second sound
Left ventricular hypertrophy
At times a systolic thrill

MITRAL STENOSIS (See Fig. 2)

Area of greatest intensity A little above the apex
Time: Presystolic
Transmission About one inch around its own area
Quality: Harsh and churning
Left auricular and right ventricular hypertrophy.
Presystolic thrill Murmur and thrill are often accentuated when patient lies on his left side Occasionally the murmur may be diastolic Auricular fibrillation is often associated

AORTIC STENOSIS (See Fig 3)

Area of greatest intensity Second interspace to right of sternum
Time Systolic
Transmission: Into the carotids.
Quality: Harsh.
Systolic thrill at base
Left ventricular hypertrophy
Weak aortic second sound or no sound other than the murmur Nearly always associated with some other valve defect.

AORTIC REGURGITATION (See Fig 4)

Area of greatest intensity: Second interspace to the right of sternum.
Time Diastolic.
Transmission: Down the sternum towards the apex.
Quality Soft and blowing
Greatly hypertrophied left ventricle.
Water hammer pulse
Visible pulsations in superficial arteries
Quincke's capillary pulse
The blood pressure is higher in the lower extremities than in the upper extremities

TRICUSPID REGURGITATION (See Fig 5)

Area of greatest intensity Tricuspid area.
Time. Systolic.
Transmission: Downward toward the liver
Quality Soft.
Right ventricular hypertrophy
Pulsating liver.
Distended veins.
Often edema.

TRICUSPID STENOSIS (See Fig 6)

Area of greatest intensity Tricuspid area.
Time Presystolic
Transmission Not transmitted.
Quality: Harsh.
Right auricular hypertrophy.

PULMONARY REGURGITATION (Congenital—Rare) (See Fig 7)

Area of greatest intensity Second interspace to the left of sternum.
Time Diastolic
Transmission: Left side of sternum.
Quality Soft
Right ventricular hypertrophy.
Distended veins and cyanosis

PULMONARY STENOSIS (Congenital—Rare) (See Fig. 8)

Area of greatest intensity: Second interspace to the left of sternum
Time: Systolic.

Transmission: Veins of neck and scapular region.

Quality: Harsh.

Right ventricular hypertrophy

Distended veins and cyanosis.

Double Mitral

Area of greatest intensity. A double murmur at apex.

Time Systolic at apex Presystolic above apex.

Quality. At apex soft *Above apex harsh.*

Transmission The apical murmur toward the left axilla. The one above the apex not transmitted

Thrill—the one above the apex—presystolic thrill.

Double Aortic

See-saw sound over aortic orifice: Both at aortic orifice.

Area of greatest intensity: Aortic orifice.

Time *One systolic, the other diastolic.*

Transmission The systolic into the carotid The diastolic down along the sternum.

Quality The diastolic, soft. The systolic, harsh

Mitral Regurgitation and Aortic Stenosis

Areas of greatest intensity: One at apex, the other at aortic area

Time· Both systolic

Transmission The apical murmur to the left *axilla* The basal *murmur to the carotid* arteries

Quality· The apical murmur soft, the basal harsh.

Systolic thrill at base.

Systolic thrill in 50 per cent of the cases at apex

Great left ventricular hypertrophy

Mitral Regurgitation and Aortic Regurgitation

Two murmurs· One at apex The other at aortic area.

Areas of greatest intensity: One at apex, the other at aortic area.

Time: The apical is systolic. The basal is diastolic.

Area of transmission Apical to the left axilla Basal down the sternum.

Mitral Stenosis and Aortic Stenosis

Two murmurs: One above the apex. The other in the second intercostal space to right of sternum

Areas of greatest intensity: One above apex, the other at aortic area.

Time One is presystolic in time, the other systolic in time

Transmission: The mitral not transmitted The aortic murmur to the vessel of the neck.

Quality: Both harsh, the apical somewhat harsher

Thrill Presystolic at apex, systolic at base

Mitral Stenosis and Aortic Regurgitation

Two murmurs One being a continuation of the other

Areas of greatest intensity One above apex, *the other at aortic area*

Time One above the apex, presystolic in time (Austin-Flint murmur). The other at the aortic orifice, diastolic in time.

Transmission: The aortic murmur along the sternum toward the apex The mitral is not transmitted very far.

Mitral Regurgitation and Tricuspid Regurgitation

One murmur heard· Prolonged, soft blowing

Time Systolic

Areas of greatest transmission: The mitral is loudest at apical impulse and can be followed to left axilla and beyond it, and is of harsher quality. The tricuspid is softer, heard loudest at lower part of midsternum, and is transmitted over the liver

General venous distention and enlarged pulsating liver are found with the tricuspid mumur

Mitral Stenosis and Tricuspid Regurgitation

Produces heart failure, cyanosis and edema.

Two murmurs are heard.

Areas of greatest intensity One over apical area, the other over lower part of sternum

Time· The mitral above the apex, presystolic in time The tricuspid at lower part of sternum, systolic in time

Transmission Mitral not transmitted. Tricuspid over liver.

Quality· Mitral, harsh Tricuspid, soft

MITRAL REGURGITATION AND AORTIC STENOSIS AND REGURGITATION

Three murmurs.

Areas of greatest intensity and time: Mitral, *at apex systolic in time and transmitted to* the left axilla. Aortic, double murmur at aortic orifice, systolic and diastolic in time

Transmission: The systolic is transmitted to the carotids and the diastolic downward along the sternum

There may be many combinations of murmurs, each one of which can be isolated in the manner described above

Conditions Influencing Organic Murmurs: The stronger the heart muscle during valvular disease, the louder is the murmur; as soon as the heart becomes weak, the murmur is less loud, and may disappear in extreme myocardial weakness. As the heart muscle becomes stronger, the murmur returns.

After exercise a murmur may become louder because of the increased work upon the heart An organic murmur, particularly mitral or aortic, is heard loudest during expiration, because the heart is more exposed at that time The loudness of the murmur is no indication of the degree of valvular damage.

Compensation: As long as the heart muscle, in spite of a valvular defect, is able to carry on a proper circulation and meet all the demands made upon it by the body, we say that compensation is good. The chamber affected by the valvular defect has become larger and its walls stronger, thus being able to overcome a certain degree of inefficiency produced by the diseased valve. But when the heart muscle can no longer cope with the defect and becomes exhausted so that the circulation is interfered with, the condition is spoken of as loss or failure of *compensation.* When the tone of the heart muscle is restored and all signs of failure of compensation disappear, it indicates that compensation has been restored. As long as compensation is good, no ill effects are manifested from the presence of a cardiac murmur.

Decompensation: Failure of compensation may result from back pressure in addition to a diseased myocardium In all cases of failure of compensation the heart muscle must be diseased, otherwise the existing valvular defect would not cause the hypertrophied heart muscle to give out, excepting when an unusually severe strain is suddenly put upon it, causing acute cardiac dilatation

Severe back pressure is brought about in the following ways:

Mitral Regurgitation: During compensation the left ventricle enlarges, as does also the left auricle, because of the extra amount of blood it receives When the auricles weaken, the lungs become congested In order to overcome this congestion, the right ventricle also hypertrophies. If this chamber becomes weak, it will cause dilatation of the right ventricle and consequently tricuspid regurgitation, with all the signs of heart failure.

Mitral Stenosis During compensation the left auricle becomes enlarged because of obstruction to the outflow of blood When this chamber weakens, blood accumulates in the lungs; to overcome this intrapulmonary pressure, the right ventricle hypertrophies. The constant strain upon the right ventricle ultimately causes it to dilate and this produces tricuspid regurgitation with signs of heart failure.

Aortic Stenosis: During compensation the left ventricle hypertrophies in order to overcome aortic resistance Compensation begins to fail as a result

of the left ventricular dilatation and this causes mitral regurgitation because the valve orifice, being overstretched, prevents the valve leaflets from approximating. This, in turn, throws more work upon the left auricle, so that it may dilate and cause pulmonary congestion, which again, may result in tricuspid regurgitation, with all the signs of failure of compensation or heart failure.

Aortic Regurgitation: During compensation the left ventricle becomes greatly hypertrophied because it has to contract upon an enormous quantity of blood. When this ventricle begins to weaken, *the mitral valve orifice dilates,* preventing proper approximation of the valve leaflet because of relative insufficiency, thus causing mitral regurgitation. This, in turn, will produce pulmonary regurgitation with congestion of the lungs. Greater intrapulmonary pressure is then productive of tricuspid enlargement, with consequent dilatation or tricuspid regurgitation, with all signs of heart failure.

It is understood, therefore, that though a person suffering from a mitral or an aortic lesion may be quite comfortable, *yet when the tricuspid and pulmonary* valves are affected, heart failure is always imminent because the right ventricle is sooner or later bound to dilate

Heart Failure: This can be defined as a condition in which the heart is no longer able to maintain the circulatory equilibrium. It may occur as a result of myocardial disease, *i. e.,* rheumatic, syphilitic, arteriosclerotic myocarditis, fatty degeneration, coronary infarction, etc., or as a result of dilatation of some of its chambers because of distention or valvular defect, endocarditis, pericarditis, etc. It may be partial — when the heart fails to respond to an added effort; or complete—when the circulation is greatly embarrassed, even when the patient is at rest.

Symptoms: Because the heart is weak, it cannot force the proper quantity of blood at the proper time through the various paths with sufficient force, and the following must result:

I. *Cyanosis,* because of insufficient oxygenation of the blood

II. *Edema* of the skin and subcutaneous tissue and often also serous effusions in the pleura, pericardium and peritoneum. (Right-sided heart failure)

III. *Dyspnea;* not enough blood is allowed to enter the lungs for aeration; stasis of unoxygenated blood in the lung produces rapid respiration, because the lung attempts to draw in as much air as possible for oxygenating purposes (Left-sided heart failure) (SEE. p. 474)

IV. *Rapid and weak heart action* If an organic murmur was previously present, it will disappear as the heart muscle becomes weaker, because there is not enough vigor in a dilated heart to drive the blood onward with sufficient force to produce the sound; as the heart grows stronger, the murmur reappears

Congenital Heart Murmurs

(SEE: Congenital Heart Disease, p. 500)

Congenital heart murmurs occur in congenital malformation of the heart valves or the great vessels that are directly concerned with the blood circulation through the heart. Since the majority of congenital heart lesions compatible with life are in the vicinity of the pulmonic orifice, murmurs produced by such lesions are audible to the left of the sternum near the base of the heart. In young children, who have no previous history of rheumatic fever or of any

acute infection and who do not present signs of left ventricular hypertrophy, when a loud murmur is heard in the pulmonic region it may as a rule be classified as a congenital murmur. Adults who have a very loud murmur at the pulmonic orifice not associated with signs of heart failure or with any murmur at any of the other orifices and who present very little left ventricular hypertrophy and who, in addition, give a history of *having* had this murmur *since* very early childhood, most likely have a congenital cardiac defect.

Pulmonary Stenosis: This is often a congenital lesion and is in the majority of cases associated with other defects, such as interauricular or intraventricular septal opening alone or the group of cardiac defects known as the Tetralogy of Fallot This quartet is comprised of (1) pulmonary stenosis, (2) defect of *the ventricular septum at the base,* (3) dextraposition of the aorta, and (4) right ventricular enlargement. This combination of lesions usually causes cyanosis. The murmur is heard over the second and third left intercostal spaces; it is systolic in time and is often accompanied by a systolic thrill The second pulmonic sound is weak or may be inaudible (SEE: Fig. 8, p. 453)

Patent Ductus Arteriosus: The ductus arteriosus, which in fetal life conducts the blood directly from the pulmonary artery to the aorta without passing through the lungs, closes soon after birth so that the blood stream is diverted to the lungs. When the ductus arteriosus remains partly open after birth, the blood does not continue to traverse the fetal course, but is diverted to the lungs in the normal way. The circulation of blood through the patent ductus arteriosus reverses itself Because the pressure in the aorta is higher than the pulmonary pressure the blood flows from the aorta into the pulmonary artery; hence there is no cyanosis The murmur thus produced is heard over the second left intercostal space as a long, loud continuous hum with increasing intensity during the systole (machinery murmur). Occasionally the murmur may be heard only during the systole It is accompanied by an accentuation of the second sound and a palpable thrill. The murmur may be transmitted to the midportion of the left scapular region.

Interventricular Septal Opening (Roger's disease): When the blood is forced by the left ventricle through the septal opening into the right ventricle (left ventricular shunt), there is no cyanosis; if because of greater hypertrophy the blood is shunted from the right ventricle to the left (right ventricular shunt) *cyanosis occurs The murmur is systolic* in time and may often be accompanied by a thrill. The murmur is usually heard over the third intercostal space near the sternum or at a point midway between the upper area of right auricular dullness and the apical impulse. Occasionally an interventricular septal defect is associated with pulmonary stenosis or with the combined lesions known as the Tetralogy of Fallot

Intraauricular Septal Opening *or* Patent Foramen Ovale: This is the most common of the congenital cardiac defects; it occurs because the foramen ovale fails to close after birth. Usually it is symptomless, occasionally it may cause paradoxical emboli. An embolus forming in a vein which is carried into the right auricle may pass through the patent interauricular septum into the left auricle and from there it may be carried through the systemic circulation and

lodge in the brain, kidney or any other organ or artery. When a murmur is produced by this lesion, it is usually very soft, occurs during the diastole and is located near the sternal edge of the third left chondrosternal articulation. The defect may occur singly or in conjunction with other cardiac defects or with other congenital anomalies.

Nonorganic or Functional Murmurs

These murmurs are also known as *hemic, anemic, dynamic*, or *accidental murmurs*. Relative insufficiency and Austin-Flint murmurs may also be classified as *nonorganic*.

A functional murmur, like an organic murmur, is of endocardial origin, but, *unlike the organic, it occurs as a result of some condition other than a defective valve*. Normally the blood is of definite specific gravity, the circulation moves at a given rate per minute, and the heart valves and the papillary muscles possess a definite degree of elasticity. Alteration in any one of these conditions may cause a slight change in the normal heart sounds.

Etiology: The actual cause of functional murmurs is still a matter of dispute. No one cause is capable of producing the various kinds of murmurs encountered. There are always at least three factors operative in the production of functional murmurs These are:

I. Insufficiency of the valve leaflets, caused by dilatation of the valve orifice.

II. Uneven tension of the papillary muscles, due either to faulty innervation or degeneration of the papillary muscles, of their tendons, or *to both conditions.*

III. Inelasticity of the valve leaflets themselves.

I. Insufficiency of the Valve Leaflets Caused by Dilatation of the Valve Orifice: This condition usually occurs in a heart whose myocardium more particularly that part of it which forms the valve orifice, is in a pathological condition.

When a severe strain, be it sudden or gradual, is brought to bear upon a defective muscle, that muscle will lose its contractility. The amount of strain required to paralyze the muscle depends entirely upon its condition. If, therefore, a weak myocardium and malnourished fibrous tissue are called upon to bear an unusual amount of pressure, they are bound to yield. As the muscle and fibrous tissue controlling the valve orifices "give way," the orifice dilates, thus causing the valve leaflets to separate, and producing an insufficiency which will persist until the heart and its fibrous tissue have regained their normal tone. But no *matter how* dilated a heart may be, so long as *that part of the myocardium which helps to* form the valve orifice retains *its normal tonicity, no murmur* will be produced.

On the other hand, though a heart may show no evidence of dilatation, if its orifice is dilated, a murmur will be audible. This form of murmur closely resembles the organic variety; it is soft and blowing in quality, though of shorter duration than is the organic, and is often transmitted a short distance along the blood stream.

Mitral Valve: A functional murmur at this valve is systolic in time. *The mitral murmur of nonorganic valvular insufficiency* may be heard either at the apex, or in the vicinity of the third intercostal space, *immediately to the left* of the sternum. It does not cause cardiac *hypertrophy, though* we should remember that a previously hypertrophied heart

may develop a nonorganic murmur. This murmur does not cause accentuation of the pulmonic second sound but this fact is not often a trustworthy sign in persons suffering from lung diseases, because, as a rule in such cases, there is an accentuation of the second pulmonic sound.

Tricuspid Valve: A functional murmur at this orifice is also systolic in time. It is much softer and of shorter duration than the mitral murmur. It is heard at the lower portion of the sternum, and is often transmitted a short distance toward the right, though not as far as the liver. The patient will be slightly cyanotic, and exertion will cause violent pulsations in the veins of the neck

Aortic Valve: At this valve the murmur is very soft, and diastolic in time; it does not cause a Corrigan's or water hammer pulse, nor capillary pulsations; neither does the diastolic blood pressure *fall to as low a level as in organic aortic* insufficiency The systolic blood pressure in the lower extremity is the same or only slightly higher than in the upper extremity When this form of functional murmur occurs in any valve the systolic blood pressure always drops from 10 to 15 or more mm after exercise. This murmur comes on as a result of a strain upon a previously weakened myocardium, it may occur in one valve as the result of a nonorganic lesion in another valve In severe dilatation several valves may be affected at the same time, and the condition may be severe enough to cause failure of compensation, giving rise to the well-defined train of symptoms known as heart failure. As soon as muscle tone is reëstablished, the hemic murmur or murmurs will disappear No murmur is heard in very severe cases of decompensation because the valve orifices are greatly dilated, causing the leaflets to remain too far apart to be of any protection to the blood stream going or coming, and also because the myocardium lacks motive power.

II. Uneven Tension of the Papillary Muscles: This may be due either to faulty innervation or degeneration of the muscles themselves, their tendinae or to both.

The papillary muscles, through the chordae tendinae, hold the mitral and tricuspid valves in a state of constant equilibrium. If for any reason either a papillary muscle or one or more of its tendinae refuse to bear their share of the burden of holding the valve leaflets at the proper tension, a very soft murmur will result. This may occur as the result of.

(*a*) Degeneration of the papillary muscle; no matter how little of the muscle is degenerated, that part cannot con*trol one or more of the tendinae;* a weakened portion in an otherwise taut valve leaflet, will permit a slight regurgitation

(*b*) Faulty innervation of the papillary muscle or of several of its tendinae, which may cause spasms or unequal contractions manifested by an uneven closure of the valvet leaflets Having, therefore, an uneven surface to guard against, the blood stream will necessarily allow a slight regurgitation of blood, which is heard as a murmur The quantity of regurgitating blood is so small that it produces no other symptoms except this very soft murmur.

This class of murmurs occurs as a rule in persons who are of a high strung or neurotic temperament and in *neurocirculatory asthenia.* The heart in such subjects is not under perfect mechanical control when enduring mental or physical strain.

Exercise will often bring out such a murmur because the extra amount of work thrown upon these muscles and tendons may excite uneven tension, the added exertion permitting a slight leak. On the other hand, exercise may cause such a murmur to disappear because under a steady strain the mechanism readjusts itself; the difference is merely a question of degree. This murmur is characterized by its extreme shortness or evanescence. It is high pitched and of a metallic whistling quality, resembling the sound produced by forcibly "swishing" a reed or stick through the air. This sound comes at the end of a fairly normal, though rapid, first sound, it is systolic in time, and occurs most frequently at the apex, in the fourth interspace to the left of the sternum, the lower part of the midsternum, or in the third intercostal space, in the order named.

Functional murmurs may either be heard more plainly when certain postures are assumed, or they may disappear altogether, depending upon the strain produced by the exertion upon the individual heart chamber and its coordinating papillary muscles.

Post-mortem: The supposedly affected valve will sometimes show no signs of loss of elasticity, but it must be remembered that after death all valves are equally inelastic. Microscopic examination may occasionally show a slight degeneration in the valve leaflets, the papillary muscles, some of its tendinae or the valve orifice.

III. Inelasticity of the Valve Leaflets Themselves: In this class of nonorganic murmurs the papillary muscles and tendons are of normal tone, and the valve orifice is not weakened or dilated; the murmur occurs as a result of inelasticity of the valve leaflets themselves Normally, the closure of the semilunar valves causes a distinct, high-pitched sound which we recognize as the second cardiac sound. Also in cases of myocarditis the valvular elements of the first sound can often be picked out from the muscular element by their high-pitched character. This high-pitched sound is caused by the closure or snap of the valve leaflets. But if the elasticity of the valve leaflets is wanting, the high-pitched snappy sound gives way to an adventitious sound, which can be recognized as a distinct murmur. It is not transmitted This variety of murmur is usually heard at the base of the heart, most often over the pulmonic orifice, and because there is no muscular element entering into the production of the second heart sound, it cannot mask the valve-leaflet sound, as is often the case in apical murmurs of this character.

Any condition that will cause loss of elasticity, either permanent or temporary, will produce an alteration of the normal sound. All forms of anemia and malnutrition, because of deficient nutrition, may cause the valves to become more or less inelastic When the valve leaflets lose their elasticity, they lack the vigor which the normal valve leaflets possess and close rather sluggishly; they cannot withstand the intracardial blood pressure; consequently, a small portion of blood leaks through the valve orifice, thus causing a faint murmur. The quantity of blood must necessarily be small; otherwise, it would produce ruptured compensation or, at least, more definite symptoms of an embarrassed circulation This murmur is not transmitted because the counter eddies set up are not strong enough to carry the sound along the blood stream. These

murmurs are systolic in time because it is the great force exerted upon the weakened inelastic valve leaflets by the systole of the heart that causes them to yield.

In some instances all the three factors mentioned as causes of nonorganic murmurs may be operative in a single case. Thus, in one patient a valve orifice may be dilated, the valve leaflets may have lost their tone and the papillary muscles may be degenerated, all from a common cause.

Austin-Flint Murmur (functional). This is a presystolic murmur heard at the apex and often occurs with aortic regurgitation It is said to be due to displacement during the diastole of the anterior cusp of the mitral valve. This acts as a partial obstruction to the flow of blood from the left auricle through the mitral valve into the left ventricle Also the peculiar position of the mitral cusp causes it to project into a double blood stream (the normal blood from the ventricle into the aorta, and the opposite or return flow from the leaky valve), thus causing vibration.

This murmur differs from true mitral stenosis by the lack of a systolic shock and its weakened intensity, as well as by its constant association with aortic regurgitation and by its time, which is early diastolic.

Graham-Steele Murmur: This is a diastolic murmur heard over the pulmonic orifice. It often accompanies mitral stenosis.

Characteristics of Functional Murmurs: 1. Systolic in time in a vast majority of the cases

2. Most commonly heard at the pulmonic orifice or over the midsternal line and third rib. Next in frequency over the tricuspid and mitral areas; rarely over the aortic.

3. Rarely transmitted beyond a short distance

4 Usually soft and blowing in character

5. Not accompanied by cardiac hypertrophy.

6. Loudest, as a rule, at the end of inspiration because at that time the lungs are under great tension which must be met by a greater effort on the part of the pulmonic valve.

7. Evanescent in character, they may disappear and reappear at various times.

8. Usually associated with some form of anemia and myocarditis.

9. When the patient improves, the murmur disappears.

Table Differentiating Organic from Functional Murmurs

Organic	*Functional*
TIME	
May be systolic, presystolic and diastolic	Usually systolic.
MAXIMUM INTENSITY	
May be heard at any one of the valve orifices	Most common at the pulmonic and mitral orifices.
AREA OF TRANSMISSION	
Each murmur heard at a certain valve has its definite area of transmission	As a rule not transmitted and very seldom beyond the precordial area
QUALITY	
Either rough and churning, or loud and blowing.	Soft, blowing

Organic		*Functional*
	DURATION	
Occupies nearly the whole of the systole, diastole or presystole.		Very short.
	HYPERTROPHY	
Cardiac hypertrophy		No hypertrophy, unless preëxisting.
	RESPIRATORY INFLUENCE	
Heard loudest *during expiration*		*Heard loudest during inspiration*
Definite history of preëxisting disease, no improvement of murmur		Anemia, murmur disappears after improvement
Signs of circulatory stasis		No circulatory stasis.

Musical Murmurs

Under the term of *musical murmurs* are included all organic and functional murmurs, which have a metallic, whistling or sonorous quailty. Most musical murmurs occur at the aortic orifice and at times also at the mitral and tricuspid valves. They are, in the majority of instances, of organic origin

Etiology: The causes of musical murmurs are many· A sclerotic valve; hardening of a projecting valve cusp, fibrous bands stretched across heart chambers near the valve orifice; a moderator band, or any other condition that will *possibly produce an added* vibration to the blood column during its course through the heart

Extra Cardiac Sounds

Cardiopulmonary or Cardiorespiratory Murmurs: In some instances a soft, exceedingly short, blowing sound which consists of a number of short whiffs not unlike an interrupted breath sound, is heard at the apex, or below the left scapular angle This sound is not transmitted; it becomes louder during inspiration and during ventricular systole; it often disappears under strong pressure with the stethoscope; it also has a peculiar superficial quality.

This murmur may be caused by the rhythmical impact of the heart against a portion of the lung covering the heart (the *lingula pulmonis*), and *may be* found in conditions where that portion of the lung becomes emphysematous or *when it is bound down by adhesions.*

Pericardial Friction Sounds: Normally the heart is so perfectly lubricated as to function noiselessly in the pericardial sac In diseased conditions of the pericardium, inflammatory exudates may cause dryness or roughening of the surfaces, thus producing a rough, grating or grazing sound, not unlike the pleural friction rub.

Characteristics: A pericardial sound is usually heard over the body of the heart or near the great vessels, seldom at the apex, and as a rule, in *the third and fourth interspaces* anteriorly. It is circumscribed in character, having no definite area of transmission *Ordinarily heard as a to-and-fro friction* sound, it may occur at any time of the heart's cycle; its rhythm, however, is not constant. It may be heard a few seconds with the systole, then with the diastole and again a little later during both; therefore, the time may vary in accordance with change of posture or the quantity of fluid present

The sound is of a rubbing quality, appearing to be superficial, and becoming louder during pressure with the stethoscope or when the patient bends forward. It is found in rheumatic, tuber-

culous, uremic and other types of plastic pericarditis, also in certain types of myocarditis such as occur in coronary thrombosis.

bronchial breathing are elicited in the left scapular region near the inferior angle when the patient lies on his left side or sits upright. These disappear when the patient assumes the prone posture. This sign is prominent in large pericardial effusions, particularly of the rheumatic type A greatly enlarged heart, especially when associated with pulmonary compression, may also present this sign.

Differential Table Between Endocardial Murmurs and Pericardial Friction Sounds

Endocardial Murmurs	Pericardial Friction Sounds
Occur constantly at a certain time of the heart's cycle.	May occur at different times in the course of a few minutes
Systolic, diastolic or presystolic	Usually to-and-fro but may occur at any time.
Heard over a valve orifice.	Heard over the body of the heart, at third or fourth interspaces.
As a rule transmitted.	Never transmitted, no venous hum
Of blowing or churning qualities.	Rubbing or grating quality
Accompanied by other evidence of murmur.	Accompanied by severe retrosternal pain
Sound is deep seated, not influenced by pressure or posture	Sound very superficial, influenced by pressure of the stethoscope and by posture

Pericardial Splashing Sound: This, when present, is heard as a distinct splashing sound synchronous with the heart action. It may be caused by a hydro- or pyopneumopericardium, and by the presence of a large pulmonary cavity half filled with fluid adjacent to the heart. At times it may be heard as a result of a greatly inflated stomach. but in this condition the sounds are of a distinctly amphoric or metallic quality Pleuropericardial friction sounds have been discussed in the previous chapter and can readily be distinguished from endocardial murmurs.

Subphrenic Friction: This is a rubbing, grating sound which can be heard at the lower part of the sternum in the infrachondral space; it is synchronous with the heart's action

Bamberger's and Ewart's Sign in Pericardial Effusion: Dullness and

The Seagull Murmur: This is a high-pitched systolic murmur having a peculiar quality, resembling the cry of a sea gull during flight while feeding. This murmur may be heard over the mitral valve or over the body of the heart. It may be congenital or acquired and is usually due to a moderator band stretched across the cavity of the left ventricle. The dislodgement of one of the tendinae so that its free end becomes adherent to the wall opposite its attachment may cause this type of murmur It may also be produced by calcareous infiltration of the free edge of a valve leaflet

CHAPTER XVII

Diseases of the Heart

The pathologic states encountered in the cardiovascular system may be the result of general systemic affection or of local disease of any of the organs comprising the circulatory system. Many diseases have a predilection for or leave their imprint upon the heart or the blood vessels or upon both so that disease of the circulatory organs results from disease elsewhere. There are also conditions in which the heart or the blood vessels are the primary diseased structures and because of their malfunction *the individual as a whole is affected, and* may present one or several of a group *of symptoms associated with cardiac* affections.

Symptomatology of Cardiovascular Diseases

The nine important symptoms associated with disease of the circulatory *system are: (1) Dyspnea; (2)* cyanosis; (3) edema; (4) pain; (5) digestive disturbances; *(6) cough; (7) palpitation*, (8) fatigability, and (9) cerebral manifestations. *The severity of any of these* symptoms and their manner of occur*rence depend upon the structures* affected and the severity of the affection.

(1) **Dyspnea**: Normally shortness of breath occurs after exertion, during certain emotional states, and because of the lack of oxygen in the respired air. This type of acceleration of the respiratory rate is not dyspnea. The respiratory rate slows down and becomes normal after a short period of rest, when the emotional state is over and when the oxygen content of the air has been replenished.

Dyspnea means rapid and labored breathing. It occurs in certain pathologic conditions, such as pronounced diseases of the lungs, bronchial stenosis, severe fevers, and heart disease. One of the earliest manifestations of impaired cardiac capacity is dyspnea on relatively mild exertion. In more advanced cases, the dyspnea on mild exertion becomes more severe and continues for some time after the exertion is discontinued. This is particularly noted in left-sided heart failure. In severe cases of heart failure, dyspnea occurs even when the patient is at rest. In bilateral heart failure, the dyspnea gives way to orthopnea, that is *the patient is unable to breathe except in* an upright position. Dyspnea is an early *symptom in left-sided heart failure. "Car*diac asthma" and paroxysmal dyspnea are *associated with advanced myocardial* failure. The patient is usually awakened *with severe dyspnea during the early* hours of the morning or at any other *time so that he is obliged to sit up.* The dyspnea may occur both during exertion and while at rest. It is accompanied by *a wheezing in the chest,* by a short, hacking cough, by expectoration of frothy bloodstained fluid, and by pulmonary edema. These episodes may occur nightly or several times a week or at longer intervals. The frequent recurrence of these attacks is a bad prognostic omen. Cheyne-Stokes breathing, if of cardiac origin, is associated with arteriosclerotic and hypertensive myocardial failure. The administration of morphine, *chloral or* other hypnotics in such cases aggravates or produces *this type of breathing.*

(2) **Cyanosis**: Cyanosis of cardiac origin *affecting the lips, fingernails* and, in more severe cases, the rest of the body is found in certain types of congenital heart disease. If this symptom

develops in other types of heart disease, it is an indication of right ventricular heart failure Cyanosis may be the forerunner of edema and may later be associated with dyspnea and other signs of heart failure.

(3) **Edema:** This is among the first symptoms of right-sided heart failure. At first the edema occurs over the feet and ankles and is seen at night; it usually disappears by morning after a night's rest. As the heart failure progresses, the edema becomes more marked and gradually ascends so that it may involve the whole body and is not remedied sufficiently by rest in bed. Associated with the edema there may develop ascites, pleural effusion, pericardial effusion, enlargement of the liver and passive congestion in other organs

(4) **Pain:** Many serious types of cardiovascular disease are not accompanied by pain. The occurrence of pain in the precordium or along the arterial or venous route, if of cardiac or vascular origin, is an indication of great interference with the circulation of blood to the affected part Precordial sensitivity, fullness or distress may occur reflexly from gastrointestinal, hepatic or pancreatic disease, or from mediastinal crowding In aortic disease, mitral stenosis, pericarditis and aortic aneurysm the pain may be paroxysmal. In the so-called cardiac neurosis, in effort syndrome, in neurocirculatory asthenia and in overindulgence in tobacco, precordial distress is brought on by exertion or excitement In angina pectoris the severe pain is usually brought on by exertion; occasionally it occurs without apparent exertion Coronary sclerosis and aortalgia may cause precordial pain on physical and mental excitement, or, when at rest, coronary infarction causes sudden severe and prolonged pain In vascular disease, pain may occur at various sites as a result of embolism, thrombosis, or obliteration This may lead to hyperemia, anemia or to gangrene of the affected part

(5) **Digestive disturbances** of cardiovascular disease are generally due to passive congestion of the digestive organs and the liver

(6) **Cough** generally results from passive congestion of the lungs; it is seen in pulmonary edema, and also when the lungs or mediastinum are crowded by a large auricle, dilated ventricles, cardiac aneurysm or aortic aneurysm Cough also occurs with dyspnea or orthopnea of cardiac origin and is often associated with mitral stenosis, congenital heart disease, and occasionally a short hacking cough accompanies or follows the pauses in ventricular extrasystoles.

(7) **Palpitation:** Palpitation may occur because of disease of the myocardium, endocardium, pericardium and also because of vascular disease and disease of the blood The rapid heart rate in these instances is due to circulatory insufficiency. Cardiac palpitation is also brought on by physical and psychic excitement, by certain drugs, and it may be caused by shock, fevers, etc Occasionally the patient may complain of cardiac palpitation when none exists, the forceful heartbeats are mistaken for a rapid rate (SEE· *Tachycardia,* p 510).

(8) **Fatigability:** Lack of endurance and a feeling of exhaustion whether at rest or with mild exertion is a frequent complaint in those having low blood pressure, in neurocirculatory asthenia, and in vasovagal disturbance. At times this is accompanied by dizziness, weakness, precordial discomfort and oc-

casionally by syncope. Fatigability is also an early sign in all types of heart disease.

(9) **Cerebral Manifestations** such as headache, faintness, confusion and forgetfulness occur in arteriosclerosis of the cerebral vessels and in hypertension. Occlusion of cerebral vessels by thrombi or by emboli may lead to hemiplegia or other types of paralysis. Syncope and, at times, convulsions may occur in heart block (Stokes-Adams syndrome). Psychosis is not of infrequent occurrence in cardiac decompensation.

Acquired Diseases of the Heart

The heart is composed of three layers of structures, the pericardium, the myocardium and the endocardium. Inflammation of the pericardium is known as *pericarditis*; *inflammation* of the myocardium as myocarditis; and inflammation of the endocardium as endocarditis When the valvular portion of the endocardium is affected it is often spoken of as valvulitis When all structures are affected it is designated by the term pancarditis or carditis Because of the intimacy of the three layers, disease in one will eventually affect its adjacent *structure or all three may simultaneously* become diseased. Thus when the pericardium becomes affected myocarditis follows; or when the myocardium is primarily affected, the pericardium, the endocardium or both may become diseased; and when the endocardium becomes pathologic first, myocarditis or pancarditis may follow.

Diseases of the heart may be congenital or acquired. *Congenital diseases* are comparatively rare. Acquired heart disease may be functional or organic.

Functional heart affections are generally caused by disease elsewhere and as soon as the underlying cause is remedied the heart's action returns to normal because structurally the heart was unaffected.

Organic heart disease denotes permanent injury to *the heart from which it* cannot fully recover. Among the diseases responsible for organic heart disease, rheumatism heads the list. Other infections such as syphilis, bacterial infections, acute contagious diseases. various systemic affections (such as arteriosclerosis), diabetes, obesity, thyrotoxicosis, nephritis, and also strain, malnutrition, poisons and toxic substances all contribute their share in causing heart affections.

Diseases of the Pericardium

Normally between the visceral and *parietal layers of the pericardium there* is a small quantity of fluid which acts as a lubricant, thus permitting free action of the heart. Because of disease or infection this exudate may undergo various changes. The exudate may become plastic or fibrinous, causing adhesions between the two peri*cardial surfaces, or between* the pericardium and adjacent structures; or the pericardium may become *thickened and calcified.* In other instances effusions of various types and degrees may develop. The effusions may consist of serum (serous pericarditis), of pus (pyopericarditis), of blood (hemopericarditis), or of air (pneumopericarditis).

The etiology of pericarditis is varied; the commonest causes are: (*1*) Rheumatic fever; (2) tuberculosis; (3) pneumania; (4) chronic nephritis; (5) cor*onary occlusion*; (6) bacterial infections

such as streptococci, staphylococci, gonococci and other infections by way of the circulation or by extension from adjacent diseased tissue, and (7) trauma, either external injuries or internal injuries by a fractured rib, the tearing away of pleuropericardial adhesions or the breaking through of a mediastinal abscess or lung abscess or a malignant growth.

Four forms of pericarditis can be recognized by physical signs:

Dry, plastic or fibrinous pericarditis

Effusions in the pericardium (pericarditis with effusions)

Pericardial adhesions (adhesive pericarditis).

Pneumopericarditis (air in the pericardial sac).



Fig 1—Acute pericarditis

Dry, Plastic or Fibrinous Pericarditis: In this form of pericarditis the acute stage is manifested by congestion, with overfilling of the blood vessels after which the layers of the pericardium become dry and sticky. As the disease progresses, the surfaces are covered with a thick tenacious exudate, or are roughened by fibrous adhesions, giving it the so-called bread and butter appearance. The pain may be referred to the left shoulder and down the arm, thus resembling angina pectoris.

Physical Signs: Inspection is usually negative so far as the precordial area

is concerned. *Palpation* may reveal a to-and-fro friction rub synchronous with the apex beat; but this friction rub is not constant and may be felt at various places, particularly at the apex of the heart or at the base. The affected area is usually circumscribed and small. *Percussion* shows no change in the area of cardiac dullness. *Auscultation* yields a superficial *to-and-fro friction sound* which is brought out more clearly by pressure with the stethoscope or the ear, and can be heard either at the apex or in the third intercostal space, and at times a little above it



Fig 2—Large pericardial effusion; note globular shadow

Effusions in the Pericardium *(Pericardial Effusions)*: Effusions may be serous, serofibrinous, *purulent* or hemorrhagic.

Symptoms often depend upon the underlying cause: *A simple serous effusion*, if not very large, will give rise to no symptoms. A large effusion will cause dyspnea, precordial fullness and definite physical signs. A pyopericardium will give symptoms of sepsis in addition to *physical signs*

Physical Signs of pericarditis with effusion depend largely upon the amount of effusion and its character

Inspection: If the effusion is large, the patient will be dyspneic, and have to assume an erect or sitting posture. The apex beat will be visible in the third

or fourth left intercostal space, near the anterior axillary line, or beyond it. If pleuropericardial adhesions precede the effusion, the apex may not be displaced by the fluid, and the left lung may be compressed. When the effusion is large there will be cyanosis and distention of the vessels of the neck and of the upper chest.

Palpation: This confirms inspection as to the extent of the apex beat. Before the effusion becomes large, a friction rub may at times be felt over the base of the heart. As the amount of effusion increases the friction rub disappears, often reappearing when the effusion is nearly absorbed. In large effusions the pulse is of low pressure and may be obliterated during deep inspiration

Percussion: This shows the area of cardiac dullness to be inverted, *i. e.*, the base of dullness is downward and the apex is upward Dullness is elicited in the fifth interspace, to the right of the sternum (Rotch's sign); shifting dullness may be elicited by placing the patient in the knee chest position. In this position, because of gravity a large area of dullness is elicited over the upper sternum and extends for a considerable distance to the right and left of it, depending upon the quantity of fluid present Ebstein's angle (cardiohepatic angle of clearness) is obliterated, and the area of relative dullness is diminished, the left and, to some extent, the right lung being retracted. Liver dullness may be displaced downward.

Auscultation: In large effusions the heart sounds are distant, rapid and often feeble. Respiratory sounds to the right of the sternum may be obliterated, as are also those close to the sternum on the left side Bronchial breathing and egophony may be heard below the right nipple and behind the angle of the left scapula If the effusion is very large, and the patient leans forward or assumes the knee elbow position, the dullness and bronchial breathing previously heard at the angle of the left scapula will disappear, reappearing when the erect posture is once more assumed.

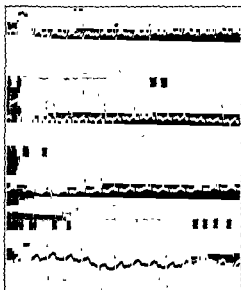


Fig 3 — Electrocardiogram showing changes in adhesive pericarditis. Note inversion of T wave in leads II and III and low amplitudes

X-ray examination will show a smooth, globular, often symmetrical enlargement of both the right and left lower borders of the heart, while the upper part of heart is narrowed

Purulent Pericarditis: This may appear as pyopericarditis or, what is commoner, as a localized collection of pus at the base of the heart, in the second or third interspace to the left of the sternum. The pus travels along the course of the great vessels. Another area favorable to local pericardial abscess is in the vicinity of the apex beat.

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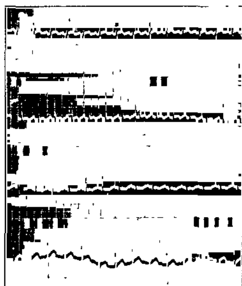


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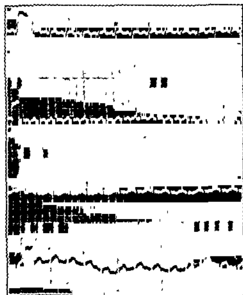


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Symptoms and Physical Signs: Symptoms are those of a septic infection and, in addition, physical signs such as local bulging, rapid heart action, and increased dullness over the site of the abscess may be elicited

Adherent Pericarditis (Chronic Adhesive Pericarditis). Adherent pericarditis is usually secondary to inflammatory disease of the pericardium. It may be limited to a few bands stretching between the two surfaces, in no way interfering with cardiac action; or again the two surfaces of the pericardium, the heart and the surrounding structures, may be united in such a way that the sac will be completely obliterated, greatly interfering with cardiac movements. This type of pericarditis is usually caused by rheumatic fever, tuberculosis or pneumonia. Pericarditis caused by cardiac infarction is usually localized.

Physical Signs: *Inspection* will usually reveal displacement of the apex beat towards the left, due to cardiac hypertrophy. Broadbent's sign, which consists of a systolic retraction on the left side in the neighborhood of the eleventh and twelfth ribs posteriorly, and Friedreich's sign, a diastolic collapse of the cervical veins, are occasionally present. The position of the apex beat does not vary with a change of the patient's posture Retraction of the apex during systole is at times observed in the presence of pericardial adhesion.



Fig. 4—Chronic obliterative tuberculosis of the pericardium (Philadelphia General Hospital)

In some instances when the heart is fixed by adhesions, the apex beat may be found in an abnormal position, *i. e.*, in the fourth interspace, higher, lower, to the left, or to the right of its normal position.

Palpation: This confirms inspection as to the position of the apex beat, and the retraction of the lower portion of the chest. The pulse may become very small during the height of inspiration (Kussmaul's pulse—pulsus paradoxus), and a diastolic shock is often felt at the apex.

Percussion: No definite *percussion* changes are demonstrable except such as may be caused by cardiac hypertrophy or dilatation.

Auscultation: There are no definite *auscultatory* signs characteristic of adherent pericarditis though there may be a systolic murmur over the mitral and tricuspid areas due to relative insufficiency

Pick's Disease (pericarditis, perihepatitis, cirrhosis and ascitis): This *is described as a condition in which the* pericardium, the mediastinum, the pleura, spleen, liver and omentum are covered with a thick white layer of inflammatory product The organs so affected look as if coated with an icing (Zuckerguss). This condition is usually, but not always, tuberculous in origin

Symptoms and Physical Signs are those of atrophic cirrhosis; ascites, enlarged superficial veins and pericardial and pleural effusions are often present The heart is not enlarged; there are no murmurs and the blood-pressure is low; there is pulsus paradoxus, and occasionally cyanosis. Because of the peculiar exudate upon the pericardium, this symptom-complex is often classified as *adherent* or *constrictive* pericarditis.

Pneumopericarditis: Gas in the pericardial sac may be due to perforation of the pericardium caused by trauma such as puncture with sharp instruments, by ulceration of the lung or the bronchi, or by an infection with gas-producing microorganisms

Symptoms: These are dyspnea, precordial distress and pain radiating to the arms and downwards along the diaphragm.



Fig 5—Chronic obliterative pericarditis with possible carcinoma of the pericardium, secondary to carcinoma of the right lung (Courtesy Dr. H. K. Mohler)

Physical Signs: On *inspection* and *palpation* the precordial area is bulging (in young individuals); the apex beat is weak or altogether absent On palpation, emphysematous crepitation may be felt

Percussion elicits tympany over the entire precordium; when the patient assumes the knee chest position a small area of cardiac dullness may be elicited near the normal apical impulse If fluid

Symptoms and Physical Signs: Symptoms are those of a septic infection and, in addition, physical signs such as local bulging, rapid heart action, and increased dullness over the site of the abscess may be elicited.

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Percussion in these cases is not of great diagnostic importance. The area of cardiac dullness may be increased because of previous hypertrophy, because of dilatation, or it may be decreased because of pulmonary emphysema.

Auscultation may reveal that the first sound of the heart resembles the second heart sound, is wanting in muscular quality, and is often high pitched, snappy and rapid (embryocardia). There may be a murmur or a friction rub, or evidence of heart block or other irregularity. The *electrocardiogram* may show alteration of the T waves and of the Q-R-S complexes.

Chronic Myocarditis: This chronic inflammation of the heart muscle is anatomically characterized by round cell infiltration of the interstitial connective tissue, followed by parenchymatous changes of the muscle fibers The myocardium as a whole may show such changes, or only circumscribed portions of it may be affected

Chronic myocarditis may be caused by: (*a*) Nephritis; (*b*) syphilis; (*c*) grave anemias; (*d*) diabetes; (*e*) rheumatic fever; (*f*) malaria; (*g*) certain wasting diseases; (*h*) toxic substances, such as lead, mercury and arsenic; (*i*) excessive use of drugs, such as alcohol and tobacco; (*j*) disease of the coronary arteries; (*k*) joint affections; (*l*) direct extension from the endocardium and pericardium; and (*m*) by arteriosclerosis

Symptoms: The most prominent symptom of chronic myocarditis is cardiac insufficiency. The heart muscle is unable to withstand ordinary strain and manifests a loss of its reserve power. During slight exertion the heart action becomes extremely rapid, the rapidity of the heart being entirely out of proportion to the exercise When a patient, who is suffering from myocarditis, rests immediately after an exercise test, the heart does not regain its previous rate for several minutes; the time required for a degenerated heart muscle to quiet down after exertion is usually two or three times as long as that needed by a normal heart. Often in cases of myocarditis the heart rate rises quickly when exertion is first begun; and when this exertion is continued beyond a certain period, the heart rate becomes slower than it was at the outset. The same holds true with the blood pressure. When blood pressure falls 10 to 20 mm of mercury during exertion, it is an indication of grave myocardial degeneration Cardiospasm, pylorospasm, colic, and angina pectoris are often prominent symptoms in this condition.

Physical Signs: On ***inspection*** the patient appears cyanosed, particularly about his finger tips, lips and ears. The apex beat may not necessarily be displaced, its position depending upon the previous condition of the heart. If the heart was previously hypertrophied, the apex beat will be displaced to the left and downward; if dilatation accompanies myocarditis, the apex beat will be displaced downward.

Palpation confirms inspection as to the location and extent of the apex beat. The pulse is weak, and arrhythmia may either be constant or induced by slight exertion Blood pressure may be very low or high

Chronic myocarditis need not necessarily change the normal *percussion* outline of the heart, but if hypertrophy or dilatation be present, the percussion changes will be characteristic of these conditions

and air are present (hydropneumopericarditis) a horizontal line of dullness can be elicited which changes in alteration of the patient's posture

Auscultation sounds depend upon the contents of the pericardial sac If only air is present in the pericardium, the heart sounds assume a loud ringing metallic quality. If air and fluid be present, a distinct splashing sound, synchronous with the heart's action, will be audible

Diseases of the Myocardium

From the standpoint of cardiac function the myocardium is the most important structure; it carries the load of the circulation A heart having no other defect except a weak myocardium will cause an inadequate circulation which will lead to heart failure

The myocardium may become hypertrophied, dilated, or, rarely, atrophied.

Heart Failure (cardiac decompensation): This may result from injury to the myocardium caused by interference with its blood supply, by various direct infections, by secondary invasion from the pericardium or endocardium, and by constant strain upon the heart muscle causing cardiac dilatation *The General Symptoms*· Weakness; diminished exercise tolerance; dyspnea; pulmonary passive congestion (basal râles, edema, cough), cyanosis, venous *distention*; enlarged liver, and edema *The Local Signs* are dilated heart, and alteration in the position of the apex beat and in the quality, force and rhythm of the heart sounds In *left ventricular failure* the early signs are pulmonary congestion (*basal râles, edema,* pleural effusion). *In right ventricular failure,* edema of the legs, cyanosis and enlargement of the liver are early signs

Acute Myocarditis: In the acute form four varieties are recognized:

1. ***Primary Acute Myocarditis:*** This is an acute interstitial inflammation of the myocardium which develops without any known definite cause. Focal infection may play a part in its etiology

2 ***Secondary Acute Myocarditis:*** This is an acute inflammation of the heart muscle which may occur during the course of some infectious disease, and may also be secondary to acute inflammation of the pericardium or endocardium.

3. ***Acute Septic Myocarditis:*** This is a localized suppurative inflammation of the heart muscle It may result from infection in some distant portion of the body, carried to the heart by the coronary arteries; or it may extend by contiguity from a suppurating pericardium or endocardium It may be caused by diphtheria, coronary occlusion and by acute infectious diseases

4. ***Rheumatic Myocarditis:*** This may be classified as a distinct entity. It is characterized by the presence of "Aschoff's bodies," general myocardial hypertrophy and often by mitral disease.

Symptoms of Acute Myocarditis: These are usually masked by the primary disease. Great weakness, cardiac palpitation with irregularity, a small feeble pulse, and dyspnea out of proportion to the underlying condition point towards affection of the myocardium

Physical Signs: *Inspection* shows the apex beat to be extremely weak, or not at all visible. A visible apex beat when palpated may be weak and slow or rapid; the pulse is weak and may be irregular; and areas of tenderness are palpable over various portions of the precordium.

Percussion in these cases is not of great diagnostic importance. The area of cardiac dullness may be increased because of previous hypertrophy, because of dilatation, or it may be decreased because of pulmonary emphysema.

Auscultation may reveal that the first sound of the heart resembles the second heart sound, is wanting in muscular quality, and is often high pitched, snappy and rapid (embryocardia). There may be a murmur or a friction rub, or evidence of heart block or other irregularity. The *electrocardiogram* may show alteration of the T waves and of the Q-R-S complexes.

Chronic Myocarditis: This chronic inflammation of the heart muscle is anatomically characterized by round cell infiltration of the interstitial connective tissue, followed by parenchymatous changes of the muscle fibers The myocardium as a whole may show such changes, or only circumscribed portions of it may be affected.

Chronic myocarditis may be caused by: (*a*) Nephritis; (*b*) syphilis; (*c*) grave anemias, (*d*) diabetes; (*e*) rheumatic fever, (*f*) malaria; (*g*) certain wasting diseases; (*h*) toxic substances, such as lead, mercury and arsenic; (*i*) excessive use of drugs, such as alcohol and tobacco; (*j*) disease of the coronary arteries; (*k*) joint affections, (*l*) direct extension from the endocardium and pericardium, and (*m*) by arteriosclerosis

Symptoms: The most prominent symptom of chronic myocarditis is cardiac insufficiency The heart muscle is unable to withstand ordinary strain and manifests a loss of its reserve power. During slight exertion the heart action becomes extremely rapid, the rapidity of the heart being entirely out of proportion to the exercise. When a patient, who is suffering from myocarditis, rests immediately after an exercise test, the heart does not regain its previous rate for several minutes; the time required for a degenerated heart muscle to quiet down after exertion is usually two or three times as long as that needed by a normal heart. Often in cases of myocarditis the heart rate rises quickly when exertion is first begun; and when this exertion is continued beyond a certain period, the heart rate becomes slower than it was at the outset. The same holds true with the blood pressure. When blood pressure falls 10 to 20 mm of mercury during exertion, it is an indication of grave myocardial degeneration. Cardiospasm, pylorospasm, colic, and angina pectoris are often prominent symptoms in this condition

Physical Signs: On *inspection* the patient appears cyanosed, particularly about his finger tips, lips and ears. The apex beat may not necessarily be displaced, its position depending upon the previous condition of the heart If the heart was previously hypertrophied, the apex beat will be displaced to the left and downward; if dilatation accompanies myocarditis, the apex beat will be displaced downward

Palpation confirms inspection as to the location and extent of the apex beat The pulse is weak, and arrhythmia may either be constant or induced by slight exertion Blood pressure may be very low or high

Chronic myocarditis need not necessarily change the normal *percussion* outline of the heart, but if hypertrophy or dilatation be present, the percussion changes will be characteristic of these conditions

Auscultation reveals a first sound that is short, feeble, and lacking in muscular quality Usually also there is a reduplication of that sound. The second sound, particularly the aortic, is accentuated When dilatation coexists, a systolic murmur will be heard at the apex and is transmitted over a small area.



Fig 6—Myocardial degeneration with cardiac dilatation

Fatty Heart: Under this heading may be included the two conditions so *prominently stressed* by older writers namely, *fatty infiltration* and *fatty degeneration*. In both conditions the heart fibers are said to be displaced or very much encroached upon by fatty tissue and this infringement necessarily weakens the myocardium, so that its normal contractile power is partially lost. The signs and symptoms of this condition are similar to those of chronic myocarditis Only a pathological examination can accurately *differentiate fatty heart* from other forms of myocardial changes.

Hypertrophy of the Heart: Hypertrophy of the heart is a physiological condition, being nature's method of en-

hancing the heart's capacity to meet the demands of the body.

The heart muscle may hypertrophy as a result of:

(*a*) Exercise.

(*b*) The effort to overcome some deficiency in one of its valves, *e.g*, mitral regurgitation (compensatory). Aortic stenosis, aortic regurgitation or a combination of these murmurs will cause left ventricular hypertrophy.

(*c*) The effort to overcome resistance in the peripheral circulation (disease of the kidney or the liver).

(*d*) Tricuspid regurgitation or other venous engorgement which may cause right ventricular hypertrophy.

(*e*) Mitral stenosis which will produce left auricular hypertrophy and right ventricular and in some instances also left ventricular hypertrophy (particularly when associated with rheumatic myocarditis) Tricuspid stenosis may cause right auricular hypertrophy.

(*f*) Increased rapidity of the circulation, *e g.*, exophthalmic goiter.

(*g*) Chronic adhesive pericarditis in which the heart may or may not be enlarged.

(*h*) Rheumatic fever, even in the absence of an endocardial lesion.

Physical Signs: The physical signs of cardiac hypertrophy depend entirely upon the amount of enlargement present and the chambers involved

In left ventricular hypertrophy *inspection* will reveal an apex beat displaced downward and toward the left; *palpation* will confirm the location of the apex beat and ascertain its increased force. The pulse is usually full, and not very easily compressible. *Percussion* will elicit an increased area of cardiac dullness If only left ventricular hypertrophy is present, dullness will be increased to the left of the sternum; if both left and right ventricular hypertrophy are present, the area of cardiac dullness will be increased to the right and left of the sternum. *Auscultation* reveals the heart sounds to be very loud and distinct; the first sound is booming in quality, while the second sound may be accentuated, depending upon the underlying cause of the hypertrophy. If the cardiac hypertrophy is caused by some intrapulmonary condition the second pulmonic sound will be accentuated, but if caused by increased systemic pressure, the second aortic sound will be accentuated.

Dilatation of the Heart: By dilatation of the heart is meant an increase in the size of the chambers of the heart due to the overstretching or degeneration of its walls. The dilatation may affect one or more chambers of the heart and may be acute or chronic.

Acute Dilatation: This is usually primary; the symptoms are those of heart failure, dyspnea, cyanosis, edema of the lungs, etc

Chronic Dilatation: This is secondary either to some valvular defect or to a gradual strain brought to bear upon a previously weakened myocardium Hypertrophy may eventually give way to dilatation, particularly in valvular disease, as the heart muscle in these cases gradually and persistently enlarges in order to overcome the deficiency of an ever-increasing leak. To compensate for this leak, the heart muscle continues to hypertrophy until it reaches its maximum, beyond that point it will dilate

Symptoms of chronic dilatation are very similar to those of acute dilatation except that the onset is more insidious

Physical signs revealed by *inspection* are cyanosis, pulsation in the jugulars,

epigastric pulsation and dyspnea; by *palpation*, edema or anasarca, downward displacement of the apical impulse, which is feeble and diffuse, and a weak, rapid and wavy pulse will be found.

Percussion shows the area of cardiac dullness to be increased in the direction of the dilated chamber Since the right ventricle is the one most frequently so affected, increased dullness is found to be downward and toward the right of the sternum.

Auscultation reveals the heart sounds to be weak, rapid, and often arrhythmic, with frequent reduplications of the first and second sounds and often functional or organic murmurs.

Atrophy of the Heart: Atrophy of the heart means diminution of the heart in weight and size Either one of its chambers or the entire heart may be so affected It is an exceedingly rare condition and may be congenital, only recognizable on x-ray examination Atrophy of the left ventricle may occur in very rare instances during the course of mitral stenosis Pulmonary tuberculosis and chronic adhesive pericarditis (Pick's disease) are associated with a small heart

Physical Signs are those of cardiac inadequacy, such as a feeble pulse, weak and irregular, often arrhythmic heart sounds and a diminished area of cardiac dullness. The E K. G. will show low amplitudes in all leads

Aneurysm of the Heart: Aneurysm of the heart is a rare condition It occurs as a sequel of ulcerative and syphilitic endocarditis and it may be due to localized myocardial degeneration or infarction as caused by coronary disease. In its chronic form it may take place in a myocardium which has undergone fibrotic changes

Physical Signs (when the aneurysm is sufficiently large): On *inspection* a pulsating area other than the apex beat is visible in the precordium; if a rib has been eroded, a pulsating tumor can be seen and felt *Percussion* may reveal an increased area of dullness corresponding to the site of the aneurysm On *auscultation* a loud indistinct murmur may be heard throughout the heart's cycle over the entire precordium An accurate positive diagnosis of cardiac aneurysm cannot be readily made by physical examination but may be revealed by the x-ray and the fluoroscope Often a positive diagnosis is only made post-mortem



Fig. 7—Aneurysm of left ventricle.

Diseases of the Endocardium

Valvular Heart Disease

Any portion of the endocardium may be the seat of inflammation, but unless the valves are affected, diagnosis of endocarditis is extremely difficult. There are three forms of endocarditis recognized: Acute, subacute and chronic.

Acute Endocarditis: Acute endocarditis is arbitrarily divided into two

classifications: (1) Simple or benign; (2) ulcerative, infective or malignant (bacterial).

Simple endocarditis is so called because as a rule this form gradually merges into the chronic form resulting in a chronic valvulitis.

Physical Signs: These depend largely upon auscultation. If the endocardium affects a valve, murmurs will be heard at that valve. Acute simple endocarditis is, in the majority of cases, due to infection, rheumatism, tonsillitis, chorea, syphilis or to the etiologic factors producing these conditions, though in many instances no definite cause is apparent

Bacterial or acute ulcerative endocarditis is an exceedingly grave condition, the majority of cases terminating in death. Those patients who may recover usually remain chronic sufferers from a badly damaged heart.

Etiology: This form of endocarditis is usually secondary to some infectious process in the body. It may occur as a result of chronic suppuration, diphtheria, scarlet fever, influenza, typhoid fever, streptococcic infection of the blood stream, gonorrhea, some suppurative processes in the bone, and rarely because of pulmonary tuberculosis

Symptoms These are irregular fever, chills, sweat, rapid loss of strength, anemia and embolic phenomena such as large spleen, large liver, joint affections, intracranial phenomena, tender sternum; and altered heart action.

Physical Signs: Inspection as a rule shows the apex beat displaced because of the rapidly increasing hypertrophy At first heaving, but as the disease progresses, the apical impulse becomes irregular and weak *Palpation* confirms inspection in regard to the position and extent of the apex beat The pulse is rapid, often irregular, and depends largely upon the heart valves affected. Thus, in mitral stenosis the pulse is small; while, *per contra,* in aortic regurgitation the pulse is large and collapses suddenly (water-hammer pulse).

In the presence of hypertrophy an increased area of cardiac dullness can be elicited by *percussion*

Auscultation will reveal a harsh murmur, usually at the mitral or at the aortic valve; often a combination of murmurs may be present.

Blood culture may reveal the infective organism

Subacute and Chronic Endocarditis:

Subacute Bacterial (Infectious) Endocarditis: This condition may develop in the absence of any previously known pathology; it may follow some local or general infection; and it may affect a previously normal valve, though more often the infection settles upon a defective valve, rheumatic or congenital The mitral valve is more often invaded, though the aortic, pulmonic, and tricuspid valves or the mural endocardium may develop vegetations or ulcerations.

Etiology: The streptococcus viridans is the etiologic factor in from 90 to 95 per cent of the cases The influenza bacillus and the gonococcus and other organisms when attenuated may affect the heart valves and run a rapid subacute course. The disease may occur at all ages but is most common between the ages of 20 and 35 years, and is somewhat more prevalent among males than among females. The organisms after entering the blood stream find lodgment in a previously damaged valve and cause the formation of vegetations; these break off and spread emboli to various parts of the body. *Chronic bacterial (infectious) en-*

docarditis is practically subacute infectious endocarditis running a longer course than usual.

Prognosis: The disease may run from three months to a year or more, depending upon the severity of the infection and the embolic spread. It is a fatal disease, though occasionally there may occur a spontaneous remission or a cure.

extremities (they are small superficial hemorrhagic spots). *Osler's nodes* and *Janeway's spots* are often found on the finger tips or palmar surfaces of the hands. The apex beat is usually forcible.

Palpation: The position of the apical impulse depends upon the amount of hypertrophy and the degree of cardiac displacement. A thrill is usually felt over the apex or base of the heart, depending upon the valve affected. Usually the lesion produced causes a stenosis of either the mitral or aortic valves, though other valves may be affected and regurgitation may accompany stenosis in the same valve or regurgitation may occur in different valves. The spleen is enlarged, often tender to touch and frequently the seat of embolic infarction, as is evidenced by sudden pain.



Fig 8—Subacute bacterial endocarditis (Philadelphia General Hospital.)

Symptoms: The patient is usually pale and weak, though he will invariably state that *he feels fine*. The temperature may range from 99° to 103° F. or higher Chills occur only rarely but sweats are common in the subacute variety. Embolic phenomena are pathognomonic. During the "Bacterial Free Stage" the temperature is normal and the blood is sterile (Emanuel Libman).

Physical Signs: Inspection usually reveals generalized pallor, and petechiae may be found on the conjunctiva or anywhere on the chest, abdomen and

Percussion: The area of dullness to be elicited will depend upon the degree of cardiac hypertrophy.

Auscultation: The murmur elicited, if at the mitral area, is at first presystolic; later it may become double. If at the aortic area, it is usually systolic, seldom diastolic. The tricuspid and pulmonary valves are infrequently affected

Diagnosis: This is usually based upon: (*a*) Endocardial rough lesion; (*b*) irregular fever; (*c*) anemia; (*d*) embolic phenomena; (*e*) petechiae; (*f*) large spleen, (*g*) sense of well being; (*h*) positive blood culture.

Chronic Valvulitis: By this term is recognized any condition that gives rise to an organic heart murmur. The symptoms and signs of chronic valvulitis depend chiefly upon the valve affected (mitral, aortic or any other valve), the condition of the heart muscle, the amount of strain upon the heart, and the presence or absence of intercurrent diseases

Mitral Regurgitation: The lesion causing mitral regurgitation is the most common of all organic valvular defects

Etiology: In the young it usually follows acute articular rheumatism, chorea, tonsillitis, and, less frequently, any one of the acute infectious diseases. In the aged the commonest cause is arteriosclerosis.

Pathology Mitral insufficiency is the result of insufficient closure of the mitral valve during ventricular systole, thereby permitting a regurgitation. The insufficient closure of the valve may be caused by contraction of the so-called "valve leaflets," permanent overstretching of the valve orifice, or by constriction of the papillary muscle and chordae tendinae, thus preventing complete approximation of the valve.



Fig 9—Chronic sclerotic endocarditis

Symptoms. During compensation there are no symptoms except that the patient may notice that he tires on exertion sooner than do some of his friends or than he previously did When compensation begins to fail, the severity of the symptoms depends entirely upon the degree of failure of compensation, rang-

ing from dyspnea on exertion to anasarca, orthopnea and cyanosis.

Physical Signs· Inspection: During compensation, general inspection is negative; the apex beat is displaced downwards and to the left, the amount of displacement depending upon the degree of cardiac hypertrophy.

After compensation begins to fail, in the early stages, when the left ventricle is still able to maintain some control



Fig 10—Mitral regurgitation (Jefferson Hospital Laboratory.)

of the circulation with the assistance of the right ventricle, the following are noted: Moderate dyspnea; rapid, weak and displaced apex beat; epigastric pulsation, pulsation at the root of the neck; slight cyanosis of the lips and finger tips; and pretibial edema at night All these become aggravated on exertion.

After failure of compensation edema and anasarca, dyspnea, feeble apical impulse displaced downward and to the left, and violent venous pulsation are noted when the patient is at rest.

Palpation. During compensation the apex beat is palpable, a little to left of the normal position and may be strong; a systolic thrill is felt in many cases.

After compensation begins to fail the apex beat is more rapid; a thrill is seldom felt; there is considerable pretibial edema, most marked at night after the patient has been on his feet all day. The pulse is rapid, and somewhat irregular as to volume. Exertion aggravates these signs.

After failure of compensation, the apex beat is weak and rapid; anasarca is well marked. The pulse may be irregular because of auricular fibrillation. Systolic blood pressure falls after slight exertion

Percussion During compensation moderate cardiac hypertrophy of the left ventricles is elicited; as *compensation begins to fail* the percussion dullness increases on both sides of the sternum.

After failure of compensation percussion reveals marked dilatation of both ventricles and the left auricle. Pleural effusion, ascites and enlarged liver may at times be demonstrated.

Auscultation: A systolic murmur, blowing in character, is heard at the apex. This may occur with the first sound of the heart, or the first sound may end with the blowing murmur.

and in severe cases the murmur may entirely displace the first heart sound. The loudness of the murmur is no indication as to the amount of leakage. The stronger the heart muscle, everything else being equal, the louder is the murmur. When the heart begins to weaken, the murmur becomes fainter Exercise always brings it out more clearly, as does also cardiac stimulation The murmur is transmitted to the left axilla, and often as far as the scapular angle

The pulmonic second sound is accentuated because of increased pulmonary pressure; and at times a reduplication of the second sound may be heard at the base. When the pulmonic second sound begins to weaken it is an indication of left auricular weakness Dyspnea and signs of pulmonary congestion are present.



Fig 11—Mitral stenosis

***Mitral Stenosis*·** This condition is second in frequency to mitral regurgitation. It is found more frequently in children and young adults; as one ad-

vances in years other cardiac lesions accompany or displace it. Women are said to be more frequently affected than men. In the early stages, when compensation is maintained, the presence of a mitral stenotic murmur is often overlooked. This has been demonstrated on a large scale in the examination of drafted men when they first entered training camps, and also when they were examined to be "mustered out," after having served in the army from 6 to 18 months or longer.

In the routine heart examination of our soldiers, frequently when the stethoscope was first placed over the mitral area, no murmur was audible, only a *very strongly accentuated first sound* being heard over the apex. But when such a soldier was placed in the recumbent posture or on his left side for one or two minutes, a distinct *presystolic thrill and murmur were often* easily demonstrated at the apex. The very fact that the military camp examiners found more mitral stenotic than mitral regurgitant murmurs among the troops, proves how easily these early cases of mitral stenosis may be overlooked.



Fig. 12—Heart showing buttonhole valve mitral stenosis (Jefferson Hospital Laboratory Da Costa, W. H Saunders Co)

Etiology: The most prominent factor in causing this form of endocarditis is rheumatism and its associated diseases, *viz*, tonsillitis and chorea, or conditions predisposing to them, and also bacterial infections. Mitral stenosis usually develops slowly. Acute endocarditis causing mitral stenosis is not very frequently found in adults past middle age. Mitral stenosis may be brought about by the same conditions that cause generalized arteriosclerosis, and may also be associated with chronic nephritis, gout and rarely with syphilis. In acute vegetative endocarditis, vegetations form on the *free margins* of the leaflets, thus causing obstruction and, in time, shrinking.

Pathology: The valvular orifice may be either "buttonhole"-shaped or funnel-shaped. The buttonhole orifice is caused by shrinking and puckering of the valve cusps because of fibrosis, and is as a rule a chronic process. The funnel-shaped orifice is usually a result of acute endocarditis; it may be brought about by adhesion of the adjacent valve

cusps. In mitral stenosis there is an obstruction to the flow of blood as it leaves the left auricle for the left ventricle; in order to overcome the obstruction, the left auricle hypertrophies Dilatation, however, occurs early in the disease because of the thin musculature of this chamber. This soon produces an overfilling of the pulmonary vein, with its resultant increased intrapulmonary pressure. The increased intrapulmonary pressure in its turn throws an added burden upon the right ventricle. As long as the right ventricle keeps its vigor, compensation is maintained; but as soon as the right ventricle begins to dilate, failure of compensation takes place. Mitral stenosis is often accompanied by mitral regurgitation

Symptoms The subjective symptoms of mitral stenosis depend upon the stage of the disease When compensation is maintained, no symptoms are complained of by the patient, except those of early dyspnea and cardiac palpitation on exertion, frequently accompanied by cyanosis. When compensation begins to fail, pulmonary hemorrhage due to pulmonary congestion is fairly common, and auricular fibrillation comes on comparatively early Congestion and enlargement of the liver and ascites are commoner than dropsy of the extremities, and embolism occurs more frequently in mitral stenosis than in any other lesion Hoarseness due to impingement of the left recurrent laryngeal nerve by the left auricle may be found in this disease After failure of compensation all the signs of heart failure are manifested, *i e.*, dyspnea, cyanosis, edema, anasarca, etc.

Physical Signs· On inspection during compensation nothing abnormal is noted, though in thin-chested children an impulse may be visible in the third intercostal space or higher, close to the sternum The apex beat is as a rule not displaced, unless the mitral stenotic lesion occurred after the left ventricle became hypertrophied, or when mitral stenosis and regurgitation are present at the same time. A purely mitral stenotic lesion (if such be possible) does not produce left ventricular hypertrophy because the left ventricle does not get an increased quantity of blood to contract upon, as is the case with other lesions. In mitral stenosis associated with rheumatic myocarditis cardiac hypertrophy is well marked

On palpation during compensation a presystolic thrill is felt a little above and to the right of the apex. This thrill is often present before the murmur manifests itself, and can be brought out more distinctly by placing the patient upon his left side. The apex beat is felt as a short systolic impulse or shock; occasionally a sharp impulse is also palpable in the pulmonic area. *After failure of compensation* the thrill may disappear, and an extremely irregular apex beat takes its place (auricular fibrillation)

The *pulse* is usually small and of low tension; in advanced cases, auricular fibrillation or flutter may be manifested

Percussion shows dullness slightly increased at the base; it extends higher and further to the left than the normal because of left auricular hypertrophy and dilatation of the conus arteriosus Dullness also extends further to the right of the sternum due to right ventricular hypertrophy. When left ventricular hypertrophy is present, the dullness extends to the left of the sternum.

The pathognomonic *auscultatory* sign attributed to mitral stenosis is a pre-

systolic murmur which is rough and churning in character; it is best heard at a point a little above and to right of *apical impulse and is not transmitted* This murmur is *crescendo* in character, and terminates with the systolic shock, resembling the sound *"ler-up-tup."* *Accentuation of the pulmonic second sound* is nearly always present In old cases the murmur may be purely diastolic

At times a diastolic murmur *minuendo* in character may be heard above the area of the apex beat, often followed by the characteristic presystolic murmur of a *crescendo* character. *After failure of compensation* the presystolic murmur may disappear, particularly so when auricular fibrillation supervenes, but the snappy first sound and accentuated second usually give a clue as to the nature of the affection A double second sound may be heard at the base, due to uneven tension in the semilunar valves

Differential Diagnosis Mitral stenosis may often simulate the following conditions: (*a*) Austin Flint murmur, (*b*) Graham-Steele murmur, (*c*) aneurysmal murmur; (*d*) pulmonary tuberculosis (because of hemoptysis); (*e*) congenital patent ductus arteriosus

Mitral stenosis may be *mistaken for* aneurysm when there is a coexisting paralysis of the left recurrent laryngeal nerve because of the hoarseness, brassy cough and pulsating auricle. Attention to the apical sounds will differentiate the two conditions. It may also be mistaken for pulmonary tuberculosis, because of hemoptysis and pulmonary congestion, and both conditions may occur in the same individual; but when a careful heart examination is made, *they can easily be differentiated*, and confirmatory evidence may be obtained by a consideration of the history, and such clinical manifestations as the presence or absence of fever, sputum examination and roentgenologic study In congenital patent ductus arteriosus the thrills and murmurs are *systolic in time and are felt* over the left base of the heart.

Aortic Regurgitation: The murmur of aortic regurgitation is caused by the incomplete *closure of the aortic* semilunar valves during ventricular diastole, thereby permitting the regurgitation of a portion of blood from the aorta back into *the left ventricle* during its diastole

Mitral Stenosis	Austin-Flint Murmur
Time · *Presystolic or diastolic*	*Time* Presystolic or early diastolic.
Point of maximum intensity—above apex	Point of maximum intensity—above apex
Crescendo in character.	No *crescendo* character
Systolic shock.	No systolic shock.
Accentuation second pulmonic	No accentuation of second pulmonic.
Not associated as a rule with aortic regurgitation and arterial phenomenon	*Associated* with aortic regurgitation and its arterial phenomena
Very little left ventricular hypertrophy.	Great left ventricular hypertrophy, heaving apical impulse.

Mitral Stenosis	Graham-Steele Murmur
Usually a presystolic murmur, heard a little above apex.	Diastolic murmur, heard along the left border of the sternum, due to incompetency of the pulmonary valves; at times heard in conjunction with a mitral stenotic murmur

Etiology: This murmur is found most frequently in young males and in early middle age. A number of conditions may be responsible for the development of aortic regurgitation, syphilis being the most frequent factor, as the spirocheta pallida have, in many cases, been isolated from the first portion of the aorta. Rheumatic fever is next in frequency,

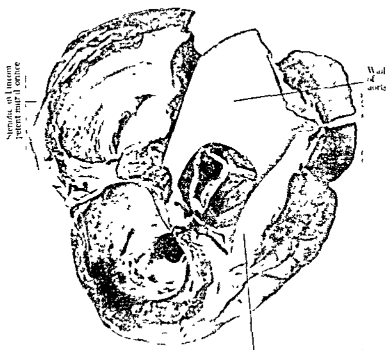


Fig 13—Aortic regurgitation and double mitral lesion (Jefferson Hospital Laboratory.)

and alcoholism, gout, pneumonia, generalized sclerotic changes, or a sudden severe strain upon a weakened endocardium likewise contribute to the production of this murmur. In children it may occur as a sequel of rheumatic fever, or of the exanthemata, but only rarely is it found as a congenital condition. Some cases have been reported in which the spirocheta have been found in the aorta (near its semilunar valve) of very young children.

Pathology: The edges of the valve segments are sclerosed, contracted or curled, thus preventing close proximity; in rare instances one of the segments may become perforated. Relative insufficiency occurs as a result of overstretching of the valve orifice. In this condition, though the valve segments are normal, because of the overstretched ring they cannot approximate; but as soon as the valve orifice strengthens and assumes its normal size, the valve leaflets approximate and the murmur disappears.

Symptoms: Aortic insufficiency may exist for a long time before it is discovered; as when compensation is maintained, and the left ventricle is not greatly hypertrophied, there are practically no symptoms perceived by the patient. In this form of endocarditis

three symptoms however stand out prominently even in the very early stage.

1. *Susceptibility of the heart to nerve* stimulation. Any excitement, physical or mental, will greatly increase the heart *rate and cause arterial pulsation in the* vessels of the neck.

2. Anemia, often causing a peculiar, *grayish, earthen appearance, associated* with cerebral anemia as evidenced by throbbing headache, dizziness, flashes *before eyes, flushes of heat and sweats.*

3. Precordial pain and oppression.

When compensation begins to fail, *dyspnea, precordial pain, aortalgia and* true or pseudo angina pectoris may occur on least exertion and excitement Insomnia and dreams become very distressing at this time. After failure of compensation, signs and symptoms of heart failure will rapidly manifest themselves

Physical Signs. Inspection reveals the following:

The *apex beat* is displaced downward and to the left, the degree of displacement depending upon the amount of left ventricular hypertrophy. In the very early stage of aortic regurgitation, very little displacement of the apex beat is noticeable, but as the condition is aggravated, the left ventricle gradually dilates and hypertrophies In well-marked cases the apex beat is often seen as a forcibly heaving impulse in the sixth interspace and left anterior axillary line and in extreme cases even beyond that point

Arterial Pulsation· Carotid pulsation is among the first visible signs of aortic regurgitation even in its earliest stage; as the disease progresses, pulsations are *seen in all the superficial arteries, in the* suprasternal notch, and in the epigastrium. In advanced cases, when the heart is greatly hypertrophied, pulsations are transmitted to the liver.

Capillary Pulse (Quincke's pulse)· When compensation is fairly well maintained—cardiac hypertrophy being well developed—a successive flushing and paling is noted in the fingernails, the mucous membranes, and over vascular *portions of the skin overlying a bony* structure, *e. g*, the forehead, scalp, malar area, etc. This phenomenon can be *brought out more clearly by applying* slight pressure over the parts, for when the hyperemia thus produced begins to *disappear, a successive waxing and waning* of a pinkish tint synchronous with the heartbeat can be noted. When *mitral regurgitation develops* as a complication, the capillary pulse often disappears, for the leakage in the mitral valve acts as a safety valve, thus to some extent reducing the arterial tension

Venous Pulse: Pulsations in the veins of the neck and other superficial veins are often noted in well-marked cases of aortic regurgitation.

Palpation: This confirms inspection as to the force, position and extent of the apex beat, and of the generalized arterial pulsations.

The *pulse* is characteristic and is *known as Corrigan's or water-hammer* or trip-hammer pulse. The impulse felt at the wrist is forcible and full but immediately recedes, leaving an empty artery; this quality can be enhanced by raising the arm above the patient's head The *blood pressure* reveals the systolic pressure to be as a rule high, 140 to *200, and the diastolic pressure very low.* usually under 60. The blood pressure in the lower extremity is nearly twice as *high as in the upper extremity.*

Percussion: This reveals an enormous hypertrophy of the left ventricle.

and often, also, of the right, and when both chambers are thus hypertrophied, the heart dullness resembles that of a pericardial effusion, However, the presence of the cardiohepatic angle of resonance (Ebstein's angle), the displacement of the forcible apex beat downward and to the left, and the throbbing



Fig 14—Aortic regurgitation Note size and shape of left ventricle.

of the arteries, easily differentiate cardiac hypertrophy from pericardial effusion.

Auscultation A diastolic murmur is heard in the aortic area at the second right intercostal space close to the sternum and is transmitted downward toward the apex Very often the diastolic murmur can be heard in the third left intercostal space close to the sternum, or over the left edge of the sternum, and at times, also in the fourth left intercostal space. When the murmur is faint, it can best be brought out by having the patient forcibly expire and hold his breath, while the examiner listens to the chest with the unaided ear.

The second aortic sound is usually weak, because the murmur displaces that sound. However, in early cases when the murmur does not occupy the entire diastolic period, an accentuated short second aortic sound may be heard which ends in a blowing murmur.

A loud systolic sound may be heard over most of the large arteries, particularly over one or both femorales; at times a double to-and-fro murmur is present (Duroziez's sign).

An associated presystolic murmur (Austin Flint murmur) is occasionally heard at the apex

There are four conditions that may cause a diastolic murmur heard at the base of the heart which should not be confounded with aortic regurgitation.

1. The soft diastolic murmur of pulmonary regurgitation is heard to the left of the sternum, and is associated with severe venous congestion and cyanosis (rare).

2. Graham Steele murmur, a diastolic murmur heard in left third or fourth intercostal space close to the sternum, and often also over the sternum is caused by overstretching of the conus arteriosus; this condition may be associated with chronic mitral disease.

3. A diastolic murmur may at times be heard at the base of the heart in exophthalmic goiter.

4. A diastolic functional murmur due to aortic relative insufficiency is audible over the aortic area. Here the cardiac hypertrophy, the characteristic radial pulse, and the capillary pulse are absent. The diastolic blood pressure is high; and the systolic pressure in the lower extremity is equal to that of the upper extremity. (Author's sign.)

fail to gain weight, have a rapid sedimentation rate and mild leukocytosis, are classed as mild manifestations of rheumatic fever since these are often the forerunners of acute articular rheumatism, chorea and of heart disease. Therefore, when the type of heart disease classified as rheumatic is found in an individual who gives no definite history of having had rheumatic fever, acute articular rheumatism or chorea, rheumatic infection cannot be excluded because he may have had one of the milder manifestations of that heterogenous group

The onset of rheumatic heart disease is usually slow and unless attention is paid to the heart during or soon after one of the rheumatic diseases its affection may be overlooked until serious and unmistakable damage has been done. Among the earliest signs is a faint systolic murmur at the apex Such a murmur in a child or young person who has rheumatic manifestations should be appraised with caution and not dismissed as a functional murmur of little importance. Often these very faint murmurs are early signs of cardiac damage

Rheumatic heart disease is a true carditis affecting the three layers of the heart but not always with the same degree of severity.

The Endocardium: The structure most commonly affected is the valvular portion of the endocardium. The mitral valve is the most frequently affected, causing mitral regurgitation or mitral stenosis, or both stenosis and regurgitation. The same lesion may be responsible for both mitral defects, which may eventually lead to heart failure or to the invasion of the affected valve by the streptococcus viridans or other organisms (SEE: p. 480). The valve next in frequency to the mitral to be affected by rheumatic disease is the aortic valve The lesion is more often an aortic stenosis and is accompanied by mitral regurgitation. Occasionally rheumatic disease may cause aortic regurgitation or a combination of aortic regurgitation and mitral regurgitation, or aortic stenosis with aortic regurgitation and mitral regurgitation, or stenosis of both the aortic and mitral valves Heart failure occurs earlier and more frequently with aortic disease than with mitral disease alone Tricuspid disease is usually secondary to mitral and aortic affection, and is rare as a primary rheumatic affection.

The Myocardium: As a primary rheumatic affection the myocardium is less frequently affected than the endocardium, but it seldom escapes secondary invasion from the endocardium and pericardium. The efficiency of the heart's action depends largely upon the integrity of the heart muscle. In rheumatic myocarditis the heart muscle becomes invaded with Aschoff's bodies, which may cause degeneration of the muscle fibers in small or large areas These in time produce either local or general cardiac dilatation, eventually leading to heart failure. The myocardium may be the primary and, rarely, the only part of the heart affected, or it may precede valvulitis. Most often the myocardial affection is secondary to the valvular infection.

The Pericardium: The pericardium is generally affected secondary to affection of the myocardium; it is rarely the primary seat of affection Occasionally all three layers may become affected simultaneously Rheumatic pericarditis is first manifested as a dry or fibrinous pericarditis which, in severe cases, may become serofibrinous, causing various

amounts of pericardial effusion. Chronic adhesive pericarditis is often a late manifestation of rheumatic pericarditis. (SEE: p. 472).

Syphilitic Heart Disease: The *Treponema pallidum* has a predilection for the root of the aorta and the aortic valve; but the ascending aorta, the arch, and occasionally portions of the descending aorta may also show evidence of syphilis. When the aortic valve is affected it causes aortic insufficiency and seldom aortic stenosis because the commissure is widened by the lesion. Aortic regurgitation originating in the adult is, in the majority of cases, due to syphilis. When aortic stenosis accompanies aortic regurgitation the etiology is usually not syphilis. The coronary arteries may be affected only at their orifices by the encroachment of intimal proliferation of the aorta; this, however, may lead to occlusion of these arteries. The syphilitic lesion in the aorta is characterized by a wrinkled and puckered appearance of the inner surface of the aorta; the lesions in the intima occur as isolated or confluent white or gray patches These lesions are responsible for the diminished elasticity of the aorta and may cause localized or diffuse aortic dilatation or aortic aneurysm. In syphilitic aortic valvulitis the commissures between the valve cusps are widened and the cusps are retracted towards the sinus of Valsalva, thus widening the orifice and causing an insufficiency but not a stenosis The myocardium may show evidence of diffuse myocarditis and cause various cardiac irregularities and early heart failure. Gumma of the myocardium may affect any portion of it When it affects the auriculoventricular bundle (bundle of His) it will produce complete heart block and may cause Stokes-Adams syndrome.

Arteriosclerotic Heart Disease: The most prevailing type of cardiac insufficiency in the aged is due to arteriosclerosis. This type may also occur in the middle aged whose arteries are hardened, and when there is hypertension. At times it may occur when the arterial tension is not very high or even when it is much lower than normal. The arteriosclerotic who has hypotension is in a more serious state than the one whose tension is moderately high Arteriosclerosis may be just an expression of old age or it may be caused by nephritis, toxic poisons or by other conditions.

The heart is usually hypertrophied, the apex beat is displaced downwards and to the left (before dilatation sets in). There is usually a loud systolic murmur at the aortic orifice accompanied by a loud ringing accentuation of the aortic second sound There may also be a loud systolic murmur at the cardiac apex, or a harsh murmur may be heard over the entire heart Cardiac irregularities such as bradycardia, extra systoles or auricular fibrillation may be heard in paroxysms, or any of these may be constant. The superficial arteries may be hard, pipestemlike or they may resemble a tendon Occasionally there is beading and tortuosity The vessels of the neck, either on the right side or both sides, may pulsate vigorously Cyanosis and dyspnea are common and attacks of angina pectoris are fairly frequent. Death may occur during an attack of angina pectoris or it may result from ventricular fibrillation, from cerebral hemorrhage, pulmonary edema or congestive heart failure

Hypertensive Heart Disease: Essential hypertensive heart disease differs

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accompany the heart symptoms. The pulse is rapid and wiry; the systolic pressure is elevated and the diastolic pressure is lowered so that the pulse pressure is fairly high In the absence of arrhythmias, the electrocardiogram usually shows a prominent P wave. When thyrotoxicosis is not controlled, heart failure will occur during a thyroid crisis. After thyroidectomy or during a remission, the heart action may return to normal unless severe myocardial damage had developed prior to successful treatment.

Hypothyroidism: A slow, sluggish heart action often accompanied by hypertension is found in myxedema. There is definite evidence of myocardial weakness The heart is pear-shaped due to dilatation and myxedematous infiltration of the musculature of both ventricles. An apical systolic murmur may occur as the result of dilatation of the mitral orifice The electrocardiogram usually shows low amplitudes of all waves. The T wave is either absent or inverted in all leads The administration of sufficient thyroid to overcome the myxedema causes a return of the T wave to its normal position on the electrocardiogram.

Angina Pectoris (Breast Pain): This term is applied to a symptom complex characterized by a sensation of pain and constriction in the chest There are two types of angina pectoris·

I. Angina pectoris associated with organic cardiovascular disease (True Angina)

II Angina pectoris independent of organic cardiovascular disease (Functional Angina).

True Angina Pectoris: Angina pectoris associated with organic cardiovascular disease is commoner than functional angina.

Etiology: The actual reason for such pain is attributed to cardiac ischemia This may be brought about by any condition that interferes with supplying an adequate amount of oxygenated blood to the myocardium for proper function. This may be due to: Coronary inadequacy resulting from coronary sclerosis; partial blocking of the mouths of the coronaries; coronary spasm; coronary emboli, and coronary occlusion Aortic disease, such as syphilitic aortitis, aneurysm of the aorta, aortic regurgitation, arteriosclerosis, syphilis, endarteritis obliterans, hypertensive arteriosclerosis, certain congenital heart lesions, pericarditis, severe anemia, and gastrointestinal disease, cholecystitis and pancreatitis may, at times, cause an attack of angina pectoris or may simulate it In the presence of any of these conditions acceleration of the heart's action causes pain. Pain of angina pectoris is brought on by: (*a*) Physical exertion, climbing, walking stairs, walking uphill, against the wind, in the cold, after a full meal, or just walking, or any other physical effort, (*b*) emotional excitement, such as anger, hilarity, anxiety, worry or brooding; (*c*) exposure to cold, taking a cold bath or washing the face with cold water; (*d*) digestive disturbance such as overeating, gastric and intestinal hyperdistention, and constipation, particularly when straining at stool; (*e*) overindulgence in tobacco and venery. Attacks of angina pectoris may develop during local infections or may follow various infectious diseases such as bacterial endocarditis, influenza, pneumonia, typhoid fever and also chronic rheumatism and gout. Occasionally no definite cause is discoverable.

Symptoms: The characteristic symptoms of an attack are: (1) Sudden on-

in many respects from arteriosclerotic heart disease, though both have many symptoms in common.

Hypertensive heart disease is brought about by the heart's effort to overcome with each systole the increased resistance in the systemic or pulmonary circulation. Whatever the cause of essential hypertension may be, whether arteriolosclerosis or the result of a hormone in the kidneys or in the adrenals, it throws an excessive load upon the heart. Therefore, as the condition progresses, the heart hypertrophies; the cerebral vessels, the coronaries and the vessels of the kidneys and of other organs are under constant strain. This often causes headache, dizziness, occasional heart consciousness, dyspnea, ringing in the ears, digestive disturbances, neurocirculatory disturbances and other signs of impaired function. Fear and apprehension are common psychic phenomena in this condition, as is angina pectoris Occasionally signs of coronary sclerosis or coronary thrombosis may develop Cerebral thrombosis may also occur and, at times, cerebral hemorrhage. Essential hypertension may eventually lead to one of four catastrophes: (1) Coronary occlusion; (2) cerebral hemorrhage, or cerebral thrombosis; (3) malignant hypertension, or (4) severe nephritis. Death may be caused by any one of these, by adrenal hemorrhage or by congestive heart failure.

Examination of the heart will reveal signs of cardiac hypertrophy; occasionally there may be a soft systolic murmur at the apex; or at the aortic region there may be a harsh systolic murmur with accentuated second aortic sound; at times there may be a diastolic murmur at the aortic valve due to relative insufficiency. The blood pressure is always high, ranging from 200 to 300 systolic and 100 to 160 diastolic. The superficial arteries are not easily compressible but are not pipestemlike or beaded. The arteries of the eye grounds (the retinal vessels) always show sclerosis Essential hypertension may develop arteriosclerosis. The electrocardiographic findings will show only left axis deviation with occasional inverted T unless arrhythmias and severe myocarditis occur as a complication Dyspnea on moderate exertion and at times while at rest is an early symptom in this condition.

Pulmonary hypertension is due to left ventricular failure; it may be caused by mitral stenosis, asthma, emphysema, pulmonary neoplasm, Ayerza's disease, congenital heart disease or various acute or chronic pulmonary affections These may throw a great strain on the right heart, causing dilatation of the right auricle and the ventricle. This may be manifested by cyanosis, dyspnea, throbbing of the veins of the neck, an enlarged and pulsating liver, general cardiac dilatation, edema of the lungs, anasarca and, finally, death by congestive heart failure

Thyroid Heart Disease: ***Hyperthyroidism:*** Tachycardia is an early symptom of thyrotoxicosis. The heart rate becomes accelerated above its usual fast rate caused by any kind of exertion or excitement, and does not readily return to its previous rate. Tachycardia persists when at rest or during sleep The first and second heart sounds are high pitched Auricular fibrillation is a common complication. A systolic murmur at the apex due to relative insufficiency may develop quite early. Dyspnea, moderate cyanosis, general weakness, sweats and tremors with either a thyroid adenoma or general enlargement of the thyroid and exophthalmus usually

ventricular or intraventricular block. The changes may be brought out several minutes after exercise.

(9) *Postparoxysmal Periods:* These may not show any evidence of change, other than the condition present before the attacks were initiated

Prognosis: Because sudden death may occur during an attack of angina pectoris, and because angina pectoris may be due to coronary occlusion, the prognosis is doubtful and depends upon its etiology. Patients with angina pectoris may live for many years and the attacks may often be controlled by rest and appropriate treatment. Angina pectoris does not occur in the presence of auricular fibrillation or other signs of myocardial failure.

Functional Angina Pectoris: This term may be applied to chest pain of the angina pectoris type occurring in persons who have no evidence of cardiovascular disease. The term functional is obviously arbitrary, because organic disease may at times be masked and therefore be considered as functional Functional angina pectoris is found among neurotic individuals, particularly if they come in contact with a case of angina pectoris; it is also found in neurocirculatory asthenia, in those leading sedentary lives who expose themselves to sudden and severe strain, and in those having digestive disturbances

Coronary Occlusion: Occlusion of one or more branches of the coronary arteries will cause sudden severe pain in the precordium. The retrosternal pain may come on suddenly and reach its height in a few minutes, or it may continue as a moderate sense of oppression with increasing severity over a period of several hours or days when it finally reaches its severe stage The pain is more often felt over the lower portion of the sternum and in the epigastrium. The pain may come on while at rest, after a meal, during severe emotional or physical strain, or it may awaken the patient from sleep. The pain is severe and agonizing and may be referred to both arms, to the interscapular regions or to the neck. In its severity it resembles that of angina pectoris or it may be either more or less severe than in angina pectoris The pain is always accompanied by severe shock When a large branch is affected death may occur instantaneously. During the attack the pulse rate is moderately rapid, about 100 per minute. The heart sounds are of poor muscle quality; there may be a gallop rhythm or a faint systolic apical murmur. Within several hours after the onset of pain, the blood pressure usually falls to a very low level If the patient survives 24 hours a friction rub due to the myocardial infarct develops over the body of the heart. The temperature rises to from 99 to 101;

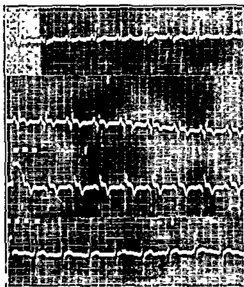


Fig. 17—Acute coronary occlusion, posterior type. (Courtesy Dr. H. K. Mohler)

set; (2) pain; (3) sense of constriction in the chest; (4) pallor; (5) sweating; (6) anxiety; (7) changes in pulse and arterial tension; (8) electrocardiographic changes; and (9) post paroxysmal changes.

(1) *Onset of the Attack*· The paroxysm comes on suddenly, usually during physical or mental exertion or after a full meal. Occasionally it comes on during sleep

(2) *Pain*· The pain is variable in its intensity It may be only a sense of uneasiness or discomfort in the sternal region, or there may be a sense of retrosternal or epigastric fullness suggesting indigestion. This may be accompanied by a sense of heaviness in the left biceps Typical paroxysms are ushered in with acute agonizing pain in the upper sternal region associated with a sense of viselike constriction. Occasionally the pain may be in the lower sternal region, the epigastrium or the umbilical region over the site of the aorta (abdominal angina) The severe pain may be referred to the left shoulder, arm and hand, or upwards to the neck as far as the angle of the jaw; or it may be referred to the right upper extremity, or to both shoulders, or posteriorly to the interscapular region Occasionally the pain may be transmitted to the lower extremities At times the pain first begins in the left shoulder and arm and then travels to the precordium. The attack of pain may last for several moments or several minutes When the attacks come on during exertion or excitement, rest, relaxation and nitrites will stop the pain When the pain occurs during sleep, sitting up or standing up out of bed will often relieve the pain, as the change of posture relieves the encroachment of the tortuous aorta upon the mouths of the coronaries. Occasionally an attack of angina is ushered in without pain (angina sina dolora) The symptoms are great anxiety of impending death, clammy sweat, dyspnea, nausea, and rapid pulse.

(3) *A Sense of Constriction in the Chest.* This usually accompanies the pain and is referred to the arm This constriction causes anxiety and fear, so that the patient is afraid to move or even to breathe During the first few moments of a severe paroxysm of viselike pain the patient may be afraid he is going to die and if the pain persists in its severity for several minutes longer he is afraid that he may not die. In other instances the sense of constriction is mild or more like distention than constriction.

(4 and 5) *Pallor and Sweats* During a severe attack the patient's face assumes an ashen gray pallor and he may sweat profusely. The skin is cold and clammy.

(6) *Anxiety*· The anxiety is proportionate to the pain, to the length of time it lasts, and the nervous make-up of the individual While the patient is always uneasy and worried during the mildest attacks, he becomes apprehensive, panicky and terror-stricken during severe attacks. In the intervals between attacks there is always anxiety and fear of the possibility of an oncoming attack.

(7) *Changes in Pulse* and *Arterial Tension:* In most instances of angina pectoris the pulse is full; it may be somewhat rapid or slow, but, as a rule, it is not altered in rate or rhythm. The blood pressure is generally elevated from 20 to 30 mm. during an attack and comes down to normal soon after the attack is over

(8) *Electrocardiogram* This may be normal. When the coronaries are affected there may be inversion of the T wave in leads I and II or evidence of auriculo-

It is to be borne in mind that the characteristics attributed to coronary thrombosis are caused by the injured myocardium resulting from the occlusion and not from intrinsic disease of the vessels. Therefore tracings similar to that obtained in anterior or posterior

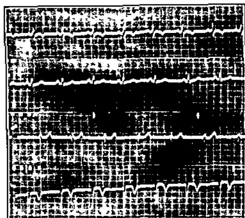


Fig 18A—Anterior occlusion Same patient as in Fig 18, six months later. (Courtesy Dr. H. K. Mohler)

coronary occlusion may be obtained in other conditions that cause injury of that part of the heart muscle which is affected by coronary thrombosis On the other hand, if a coronary branch supplying any of the silent areas of the myocardium becomes thrombosed, characteristic signs will be absent Therefore, if a patient has most of the clinical signs of coronary occlusion and fails to show any characteristic electrocardiographic tracings, he should nonetheless be treated for coronary occlusion. Typical signs of coronary occlusion do not generally develop before myocardial damage has been established; that is, 24 to 48 hours or longer after the occlusion has occurred. When cardiographic tracings are characteristic of recent occlusion, traces of occlusion remain for months or years after apparent recovery.

Prognosis: The patient may die during the first attack, or he may live for several days and die of ventricular flutter, from embolism or myocardial failure If he survives the first two weeks he may recover, but must remain in bed for six weeks or longer. Subsequent fatal attacks may occur.

Coronary occlusion may have to be differentiated from acute pancreatitis, perforating peptic ulcer, gallstone or kidney colic, and acute peritonitis of the lesser omentum

Differential Table of Coronary Occlusion and Angina Pectoris

During Attack

Coronary Occlusion	Angina Pectoris
Pain sudden, felt in lower sternum or epigastrium	Pain sudden, felt more often in upper sternal region
Pain often when at rest	Pain more often during effort
Shock	Excitement and fear, no signs of shock
Pulse feeble, rapid.	Pulse full
Blood pressure falls rapidly	Blood pressure is sustained.
Pain requires large doses of an opiate; not stopped by nitrites	Pain often stopped by nitrites (nitroglycerin under tongue or inhalation of amyl nitrite)
Duration of pain usually prolonged; may last hours or days.	Duration comparatively short, may last several minutes to half an hour.
Heart sounds feeble, may have gallop rhythm or murmur.	Heart sounds not altered, may be strong
Dyspnea and cyanosis.	No dyspnea or cyanosis

a mild leukocytosis, increased sedimentation rate and occasionally mild cerebral symptoms develop.

Electrocardiographic Changes Following Coronary Occlusion: Electrocardiographic changes often do not occur until 12 to 48 hours or later, after the first appearance of coronary symptoms. When a large portion of the myocardium is badly damaged, electrocardiographic changes occur early and remain for a long time after clinical recovery has taken place. Occasionally definite electrocardiographic changes may be absent, though all the clinical manifestations of coronary occlusion are unmistakably present.

Myocardial infarctions generally assume characteristic patterns dependent upon where the infarct occurs.

Anterior Occlusion. Lead I: High S-T take-off; flattening or inversion of T wave.

Lead II: The T wave may occasionally be flattened or inverted, or there may not be any change. The descending limb of the R wave may show splintering

Lead III· Depression of S-T interval.

Lead IV: The R wave is absent; the S-T take-off is elevated. The T wave is inverted Q1 or Q4 may be present.

Posterior Occlusion: Lead I: Depression of S-T interval

Lead II: Flattening or inversion of the T wave. Prominent Q wave.

Lead III: High take-off of S-T, and inversion of T wave. Q2 and Q3 often present.

Lead IV: No change generally. In severe infarctions T4 may be inverted.

Résumé: Anterior Occlusion: The S-T take-off is high in lead I and lead IV. and low in lead III. The T wave may be flattened or inverted in lead I and lead II and is inverted in lead IV.

Posterior Occlusion: The S-T take-off is depressed in lead I and high in lead III. The T wave is flattened or inverted in lead II and lead III; the Q wave is usually prominent in leads II and III; in lead IV, the T wave shows little change from normal or may be higher. Inverted T4 is seen in severe occlusion.

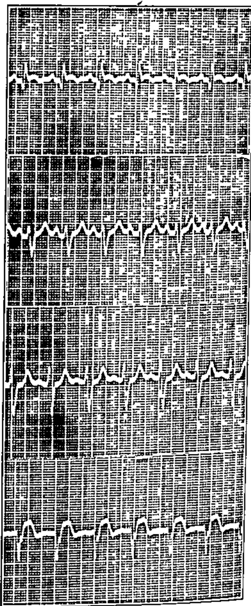


Fig. 18—Anterior occlusion (Courtesy Dr. H. K. Mohler)

other. Dr. Maud Abbott called attention to the following: When the pressure is greatest in the left ventricle, the blood will flow from the left ventricle, into the right ventricle ("arterial venous shunt"), causing no cyanosis. If, however, the pressure is greatest in the right ventricle, the blood will flow from the right ventricle into the left ventricle ("venous arterial shunt") and cause cyanosis

Congenital heart affections may therefore be divided into I. Cyanotic group (venous arterial shunt). II Noncyanotic group (arterial venous shunt)

Cyanotic Group (venous arterial shunt): Such a disturbance of the circulation may occur in several ways:

1 By direct right-to-left shunt through a ventricular septal defect, when the pressure is high in the right ventricle, as is the case in an associated pulmonary stenosis

2. By direct admixture within the chamber which may occur because of complete absence of the cardiac or arterial septum.

3. In dextroposition of the aorta, when the mouth of the aorta overrides the right ventricle, venous blood passing directly from the right ventricle into the systemic aorta Under such circumstances, the conditions are present for the development of a true congenital cyanosis



Fig 19—Congenital heart disease—pulmonary stenosis—patent foramen ovale (Philadelphia General Hospital)

Pulmonary Stenosis: This is probably the commonest of all cardiac defects. It is usually associated with a defect of the ventricular septum

Longer life is compatible with pulmonary stenosis than with any other congenital heart lesion. The chief symptoms are a palpable systolic thrill and a systolic murmur heard in the pulmonic area, accompanied by a weakened or absent second sound; often the first sound is

Coronary Occlusion	Angina Pectoris
After Paroxysm of Pain Has Stopped	
Shock	No shock as a rule.
Low blood pressure	Blood pressure may be high or unaltered from the usual.
Poor heart sounds	Heart sounds may be full and strong.
Pericardial friction rub	No pericardial friction
Leukocytosis.	No leukocytosis.
Subfebrile temperature.	Normal temperature.
Increased sedimentation rate	Normal sedimentation rate
Definite electrocardiographic changes	Electrocardiographic findings may be normal

Other Conditions Simulating Heart Pain: Substernal or epigastric pain may develop after a heavy or indigestible meal; from insulin hypoglycemia; from injection of large doses of epinephrine; and in the presence of pericardial adhesions, large pericardial effusions mediastinal tumors, plastic pleurisy, pneumothorax, emphysema, pulmonary fibrosis, mediastinitis, mediastinal urticaria, intercostal neuralgia, aortitis, aortic aneurysm, and various heart lesions associated with heart failure, pancreatitis, cholelithiasis and peptic ulcer.

Congenital Heart Disease

Congenital heart disease is relatively rare but is nonetheless of great importance. When diagnosed early, proper guidance of the individual may prolong life In many instances when the patient is an adolescent or an adult, and the previous history is not reliable, some of the congenital heart murmurs are not readily differentiated from some of the acquired murmurs.

Anomalies of Position

Dextrocardia, transposition of the heart to the right side of the sternum so that the apical impulse is in the fifth interspace, 7 to 9 cm. to the right of the sternum, may occur alone or in conjunction with situs inversus of the abdominal viscera Dextrocardia should be differentiated from lesions in the left side of the chest which push the heart to the right, or from lesions in the right chest which pull the heart to the right.

Ectopia Cordis: Other anomalies of position are ectopia cordis associated with fusion of the chest wall and of the abdomen; the heart may lie in the neck, outside of the chest wall or in the abdominal cavity.

Anomalies of Structure

The commoner structural defects in the heart, compatible with life, are defects in the interauricular or interventricular septa, defects in the pulmonic valve, retention of the ductus arteriosus, coarctation of the aorta, and congenital heart block.

Congenital heart defects interfere with the normal circulation of the blood through the heart, the great vessels or both, thereby deflecting the arterial blood into the venous channels or the venous blood into the arterial channels. When an opening occurs in the interventricular septum some leakage through that opening from one chamber into the other is to be expected. The direction of the flow will depend upon the preponderance of pressure in one chamber over the

Dilatation of the pulmonary artery is to be considered as a functional consequence of the increased pressure produced by the connection between it and the aorta or between the right and left chambers of the heart. Such dilatation may be determined by physical signs and x-ray examination. *Percussion* will yield a ribbon-shaped area of dullness in the

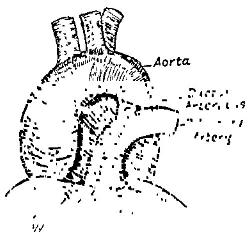


Fig 21—Patent ductus arteriosus.

first and second interspaces to the left of the sternum, Gerhardt's area of dullness *Auscultation* will reveal an accentuation of the pulmonary second sound, and the fluoroscopic and x-ray findings will show a marked bulging of the pulmonary arc The electrocardiogram is not apt to be helpful in the diagnosis of these defects unless unusual strain on the right side of the heart has caused a preponderating hypertrophy of that side. Lewis has stated that in uncomplicated patency of the ductus arteriosus the curves should be normal. Elsewhere he has mentioned exaggerated amplitude in several leads of the electrocardiogram as an important sign in congenital heart disease

The diagnosis of a congenital defect of the heart with arterial venous shunt may be based upon the following points: (*a*) A young patient with (*b*) no history of rheumatic fever or other illness commonly causing endocarditis; (*c*) absent or slight heart symptoms; (*d*) marked and peculiar murmurs and perhaps a thrill in the upper left precordium, and (*e*) good cardiac functional capacity.

1. ***Patent Ductus Arteriosus*** (Botalli): In the fetus a connection exists between the pulmonary artery and the aorta which carries practically all the blood entering the pulmonary artery into the general circulation (Fig. 21). Under normal circumstances this tube becomes closed off in the transition from fetal to extrauterine existence and undergoes atrophy If it persists as a communication, it constitutes a real danger, both from the standpoint of strain upon the heart and because of the liability of the edges and the walls of the patent duct to bacterial invasion.

Symptoms: Patent ductus arteriosus, like the other defects of the noncyanotic group, is usually symptomless, particularly in early life, and is recognized by its distinctive physical signs Frequently it is associated with other defects, and then, of course, the physical signs are modified. While the clinical picture is definite in adults, it is not so in infants, and there is often difficulty in distinguishing patent ductus arteriosus from other defects of the group. Aneurysm of the first portion of the aorta at the sinus of Valsalva, rupturing into the pulmonary artery, may simulate patent ductus

Adults with patent ductus arteriosus are usually anemic. As a rule, cyanosis is entirely absent, as is clubbing of the fingers and toes If present, cyanosis is either very slight or transitory, appearing only on exertion, or, like the

indistinct. If the stenosis is marked and is accompanied by a defect of the septum, the blood will flow from the right to the left ventricle, and then into the aorta (venous arterial *shunt*), so that the murmur will be transmitted to the aorta and to the carotids. When the ductus arteriosus has remained open, the coexistence of pulmonary stenosis may



Fig 20—Typical case of pulmonary stenosis

result in accentuation of the second pulmonic sound, while the purring murmur transmitted to the carotids, which is characteristic of the patent ductus, will also be audible.

Smith states that 69 per cent of the lesions which may occur on the right side of the heart are due to pulmonary stenosis, resulting from endocarditis during intrauterine life. The harsh, basal systolic murmur, transmitted to the clavicle, the increase of the cardiac dullness to the right, the pronounced congenital cyanosis, clubbing of the fingers, polycythemia, and splenomegaly make the diagnosis fairly easy.

Prognosis: Such patients usually die in childhood, but many reach early adult life and die, not of cardiac lesion, but of pulmonary tuberculosis. Another not infrequent termination is from bacterial endocarditis developing at the seat of the defect.

Noncyanotic Group (arterial venous shunt): This group consists of the following main defects: (1) Patent ductus arteriosus; (2) patent foramen ovale; (3) defect of the ventricular septum; (4) aortic stenosis; (5) coarctation of the aorta

The first three defects are closely related anatomically in that they represent circumscribed openings between the right and left sides of the heart (considering the aorta and pulmonary artery as continuations of the left and right ventricles). Under such circumstances the passage of blood is from the left side to the right, *i e*, arterial into venous blood, thereby exerting strain upon the right heart, but giving no cause for cyanosis, the arterial blood oxygenation being normal. However, an important feature of this condition is that a change of pressure in the lungs may convert the arterial venous shunt into a venous arterial one, with the production of transient or terminal cyanosis.

The presence of striking physical signs and the absence of symptoms are the characteristic features of this group

Physical Signs: There is a peculiar harsh murmur of unusual rhythm and intensity, often accompanied by a thrill, situated to the left of the sternum in the first or second interspace, or over the middle of the precordium, and in many instances associated with evidences of dilatation of the pulmonary artery.

Dilatation of the pulmonary artery is to be considered as a functional consequence of the increased pressure produced by the connection between it and the aorta or between the right and left chambers of the heart. Such dilatation may be determined by physical signs and x-ray examination. *Percussion* will yield a ribbon-shaped area of dullness in the

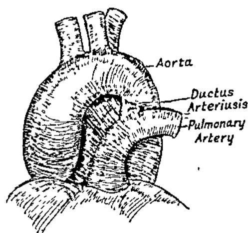


Fig. 21—Patent ductus arteriosus.

first and second interspaces to the left of the sternum, Gerhardt's area of dullness *Auscultation* will reveal an accentuation of the pulmonary second sound, and the fluoroscopic and x-ray findings will show a marked bulging of the pulmonary arc. The electrocardiogram is not apt to be helpful in the diagnosis of these defects unless unusual strain on the right side of the heart has caused a preponderating hypertrophy of that side. Lewis has stated that in uncomplicated patency of the ductus arteriosus the curves should be normal. Elsewhere he has mentioned exaggerated amplitude in several leads of the electrocardiogram as an important sign in congenital heart disease.

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form previously mentioned, may be terminal (cyanosis tardive)

Characteristic Physical Signs A peculiar rough murmur, systolic in time or continuous (machinery murmur) with maximum intensity in the pulmonary area or just beneath the left clavicle, *is the most important physical sign* This murmur is transmitted upward to-

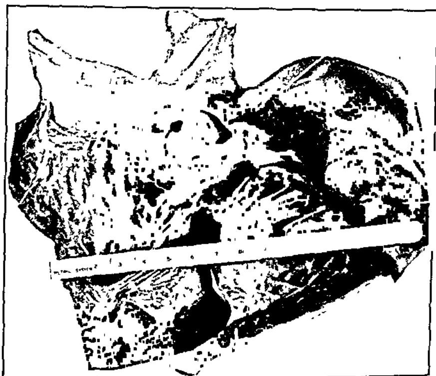


Fig 22—Aneurysm of the sinus of Valsalva

ward the left clavicle, and, depending upon its loudness, may be heard over the whole precordium and in the back. Accompanying the murmur may be a thrill of the same time, *i. e*, either systolic or continuous The pulmonary second sound is usually accentuated, and this is considered an important point in differentiating patent ductus arteriosus from pulmonary stenosis, in which the pulmonary second sound is often weak or inaudible. Dilatation of the pulmonary artery is the rule and this gives the signs already enumerated —Gerhardt's dullness and, under the fluoroscope, a prominent, actively pulsating pulmonary arc. Increased vascular hilum shadows also speak for pulmonary dilatation Laboratory aids other than the x-rays are of little value in the diagnosis of this defect.

Occasionally there is paralysis of the left vocal cord due to pressure on the left recurrent laryngeal nerve. This is also observed in association with mitral stenosis in which it has been ascribed to the direct pressure of the dilated pulmonary artery.

Patent ductus arteriosus is frequently the site of a bacterial endocarditis. The vegetations occur on the pulmonary side of the communication and may extend down the pulmonary artery and involve

the pulmonary cusps The occurrence of such a lesion is a dangerous complication, for the vegetations are easily broken off, to be carried into the lung, there to produce a suppurative bronchopneumonia

Susceptibility to infection constitutes the great danger of this lesion and is an important reason for early recognition and prophylactic care

Maud Abbott has shown that out of 67 cases of patent ductus arteriosus, 15, or 22 per cent, showed a bacterial lesion While bacterial endocarditis is usually responsible for the fatal termination, increased strain upon the heart may lead to failing compensation or to a sudden paroxysm, such as a suffocative attack, which may be responsible for death

2. ***Patent Foramen Ovale:*** This defect, which is the commonest of this group, is perhaps the least often recognized during life, due both to the fact that it is often latent and likewise because physical signs, when they do exist, are not generally correctly interpreted

Symptoms· Individuals who have a patent foramen ovale are usually of slight build, often of infantile development Although frail and delicate, they are usually harmoniously proportioned, but they may have an associated spinal curvature A very important point, possibly better mentioned with the x-ray findings, is hypoplasia of the aorta This was noted many years ago by Maud Abbott, and has been separately described by French and German observers It is a definite part of the clinical picture of patent foramen ovale. The murmur of patent foramen ovale has been described as inconstant and variable It may come and go and vary as to time, although it is usually systolic A thrill is not often associated Frequently the pulmonary artery is dilated, giving the signs already mentioned In addition, the roentgen rays may show the narrow aorta and a general enlargement of the heart, especially of the right ventricle

The first symptoms of patent foramen ovale may develop after some condition which raises the pressure in the pulmonary circuit and converts an arterial venous into a venous arterial shunt, with an attending cyanosis This may be transient and disappear when the cause of the heightened tension is removed, or may occur as a suddenly developed deep cyanosis in the course of cardiac failure or marked pulmonary disease, such as pneumonia, when it constitutes an important evidence of the presence of this defect In the latter event it is apt to persist as a terminal cyanosis Patent foramen ovale, unlike patent ductus arteriosus, is not subject to bacterial invasion; and from the standpoint of infection can be disregarded. A very curious and dangerous phenomenon has been described with patent foramen ovale; that is, a *paradoxic embolus,* perhaps arising at some distant point, passes through the foramen and enters the general circulation.

A number of cases have been mentioned in the literature in which defects of the auricular septum have been associated with idiocy. Morse, in a study of 100 cases of congenital heart disease, from his private practice, noted mental deficiency in more than 10 per cent of his cases.

3 ***Ventricular Septal Defects*** (Maladie d'Roger): Ventricular septal defects are frequently associated with other anomalies, and rarely do they occur alone They are located, most commonly, directly beneath the aortic cusps and just anterior to the unde-

fended space. The communication permits a shunt of blood from the left ventricle to the right, which has been evidenced by a patch of fibrosis or a collection of vegetations on the opposite wall of the right ventricle. However, from the standpoint of strain, this type of defect *in many instances might be* disregarded, as it interferes in no way *with perfect cardiac function* and is consistent with a long and healthy life. The importance of defects of the ventricular septum is not the strain upon the heart, as it is in anomalies of the auricular



Fig. 23—Intraäuricular septal opening (patent foramen ovale). Note widening of pulmonary conus and artery, obliteration of cardiac angle, heart enlarged to the right; aortic knob *not visualized.*

septum, but the possibility of the development of vegetative endocarditis about the edges of the defect

Symptoms· These defects, probably more than any other in the noncyanotic group, are symptomless; indeed, the French (Laubry and Pezzi) have applied to them the term "functional silence"

thus being of little functional importance, may give rise to the loudest murmurs The pulmonary second sound is present, but not accentuated as a rule Occassionally an interventricular septal defect is associated with three other defects. The quartet is known as *Tetralogy of Fallot.* The lesions are: *Pul-*



Fig 24—Congenital heart lesions (Patent foramen ovale)

They are recognized by their distinctive physical signs A harsh, even murmur filling the entire systole, accompanied by thrill in about half the cases, situated over the middle of the sternum or in the third or fourth interspace to the left of the sternal border, is the most frequent and quite often the only evidence of this defect A valuable lesson to be learned is that defects causing little or no strain upon the heart, and

monary stenosis, interventricular septal defect, right ventricular enlargement, and dextroposition of the aorta. These lesions cause cyanosis.

Prognosis In the arterial venous or *noncyanotic group, those cases with distinctive* physical signs, but excellent functional capacity as already stated, the chances are good for a long and healthy life. Two dangers exist, infection and strain. Death may occur from bacterial

stethoscope is applied to the popliteal space.) (2) There are dilated and pulsating intercostal vessels, often associated with erosion of the lower borders of the ribs; also dilatation and pulsation of the internal mammary, scapular and epigastric arteries. (3) A systolic murmur may be heard over the precordium, the interscapular region and over most of the dilated arteries that form the collateral circulation. (4) Cardiac hypertrophy occurs early. (5) The x-rays will reveal a decrease in the size of the aortic knob, or an absence of the knob, dilatation of the ascending aorta, enlargement of the left ventricle, and notching or irregularities of the lower borders of the ribs

For other anomalies of the aorta, SEE: p 526.

Functional Abnormalities (Disturbances of Rhythm)

The disturbances of rhythm may be loosely classified under three subdivisions:

I. Rapid rate with regular rhythm
II. Slow rate with regular rhythm
III. Irregular rhythm (with rapid or with slow rates).

I Rapid Rate with Regular Rhythm *(Tachycardia):* The vagus and sympathetics, while not concerned with initiating the cardiac impulse, have nevertheless a decided influence upon the heart rate The vagus slows it and the sympathetics accelerate it When the vagus is stimulated or irritated by pressure over the carotids or over the eyeballs or at any other point, or is acted upon by physostigma (eserin) or by acetyl cholin (mecholyl), the heart rate becomes slower. Also when the sympathetics become paralyzed, the vagus remains unopposed and the heart rate slows down. On the other hand, when the vagus is paralyzed by atropine, by intracranial or by intrathoracic pressure, the heart rate is accelerated because the sympathetics are unopposed; so also when the sympathetics are stimulated by drugs, toxins or in any other manner, the heart rate becomes rapid. In both vagus retardation or stimulation and in sympathetic stimulation or retardation, while the heart rate may become accelerated (tachycardia) or retarded (bradycardia), so long as the cardiac impulse originates in the sinoauricular node a regular rhythm is maintained; that is, the spacing between beats are of equal length, all being shorter than normal in tachycardia and longer than normal in bradycardia The electrocardiographic tracings will show the normal sequences of the P-R-T waves

Simple Tachycardia or Sinus Tachycardia: This may occur in cardiovascular affections, in functional disturbances, in the various neuroses, reflexly from other organs, and in fevers.

An increase in the cardiac rate may be nature's method of supplying an adequate amount of blood per unit of time In such cases, either the heart is incapable of delivering the required quantity of blood in a given time, or the blood vessels are incapable of carrying the volume of blood delivered by the normal heart, beating at a normal rhythm In either case, smaller quantities of blood are delivered at a faster rate. While the heart beats faster, the circulation in general may not be disturbed

Tachycardia may also occur because of disease of the myocardium resulting from rheumatic affections, syphilis, thy-

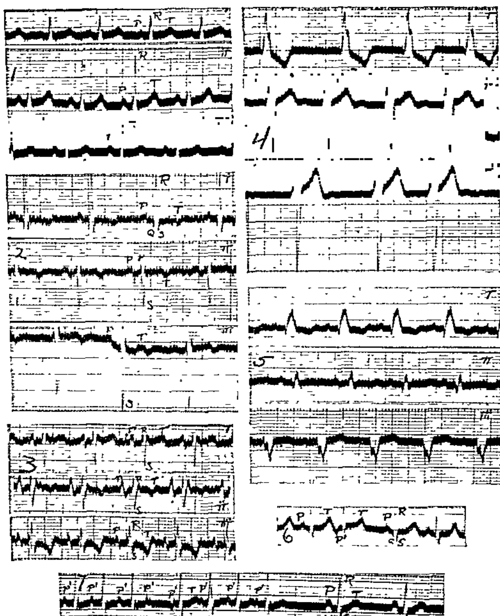


Fig 27—Normal and abnormal electrocardiograms.
(*T. M McMillan, M.D*, Philadelphia Hospital)

1 Normal heart rhythm.
2 Left ventricular preponderance and inverted T wave
3 Right ventricular preponderance
4 Complete left branch, bundle block and auricular fibrillation
5 Partial left branch bundle block.
6 Auricular extrasystole.
7 Auricular paroxysmal tachycardia

complains of palpitation while the heart rate is slow, but the force of the beat is increased; and at times palpitation is associated with precordial pain and pressure. In neurocirculatory asthenia, the heart rate may be rapid or it may become rapid as the result of emotional upset or moderate physical effort.

On the electrocardiogram, tachycardia is noted as rapid, regularly spaced, regularly recurring P-R-T waves in all leads.

Paroxysmal Tachycardia: This may occur in persons who are presumably in perfect health, and also in those who have definite myocardial damage Paroxysmal tachycardia of auricular origin is usually benign, while paroxysmal tachycardia of ventricular origin is more often an indication of serious heart damage The attacks come on suddenly, at times without any apparent provocation Excitement, toxemia and overindulgence in tobacco may be contributing factors. The attacks may last from several minutes to an hour or longer, and stop just as suddenly as they begin These paroxysms may come on once a month, once a week, more often or less frequently. During the attack, there may be some headache, dizziness and a sense of precordial oppression; the patient is conscious of the palpitation and is usually nervous and fearful. The heart rate may vary from 160 to 200 per minute, and is generally regular. In most instances the auricular rate is as fast as the ventricular. Exercise does not increase the rate, and rest does not slow it. Though paroxysmal tachycardia of auricular origin is usually benign, there are three cardiac conditions in which the accelerated cardiac rate may be serious. These are: (1) Mitral stenosis; (2) left ventricular dilatation, and (3) coronary insufficiency. In these conditions the unusually rapid heart action may cause pulmonary edema, cardiac asthma and heart failure.

A definite diagnosis as to the type of irregularity is best made by an electrocardiographic study. SEE: Fig. 27, No 7, p. 511.

As to the treatment of this type of arrhythmia, a paroxysm may occasionally be aborted by pressure exerted over the eyeballs or over the carotid sinus or by the hypodermic administration of 20 to 50 mg. of mecholyl, or two to four drachms of syrup of ipecac by mouth.

Auricular Flutter: The impulse arises from a single focus and continuously circulates at a fast rate over the same path in the auricle in the vicinity of the openings of the superior and inferior venae cavae.

In this irregularity the auricle may beat at a rate of 250 to 300 per minute, and the beats are rhythmic and uniform, while the ventricle may in comparison be rather slow and less responsive to auricular stimulation. The ventricular beats, however, are feeble and much more rapid than normal. The auricular impulses are partially blocked in their passage to the ventricles. The block may be two to one or three to one; the ventricular rate would therefore depend upon the degree of block. A two to one block would cause a faster cardiac rate than a three to one block. This condition may be recognized by the occurrence of distention and extremely rapid impulse in the jugulars, the apical impulse being feeble, at times irregular, and comparatively slow. The pulse is soft and compressible. It may be manifested in paroxysms lasting but a short time, or it may occur for quite a long period, or just before death.

Auricular flutter is usually due to myocardial degeneration or rheumatic affections and, rarely, to disease of the nervous system. The administration of large doses of digitalis and strophanthin may change the flutter to fibrillation and then to normal rhythm. Quinidine sulfate also slows the flutter. Exercise does not increase its rate nor does rest slow it. When the cardiac rate is irregular in flutter, mild exercise will often restore it to regular but rapid rhythm. On the electrocardiogram auricular flutter is characterized by a rapid heart rate and an increase in the number of P waves in relation to the R-T complexes. When there are three P waves to one R-T complex, it indicates a three to one block; if two P waves occur to each R-T complex then the block is two to one (See. Fig 28, No 11, p 512).

II **Slow Rate with Regular Rhythm:** ***Sinus Bradycardia:*** A constant slow heart rate between 50 and 60 per minute is occasionally found as an individual or family peculiarity In the aged, after fatigue, during exposure to intense cold, during convalescence from fever, in jaundice and in myxedema, the heart rate is slow. Bradycardia is also a symptom of intracranial pressure due to hemorrhage or tumor. In meningitis, typhoid fever, severe myocarditis, in certain types of arteriosclerosis, in asphyxia and anoxemia, the heart rate is definitely slowed down Bradycardia may also be produced by certain drugs, such as digitalis, opium, aconite and acetanilid or other coal-tar derivatives and by various poisons Stimulation or irritation of the vagus or blocking of the sympathetics are other causes of reduced bradycardia. In these conditions the electrocardiogram shows normal P-R-T sequences with a lengthening of the diastolic phase. Bradycardia developing in one whose cardiac rate has previously been normal, accelerated or irregular should suggest the possibility of heart block.

Heart Block: This results from interference with the normal conduction of the impulse which may be blocked any-

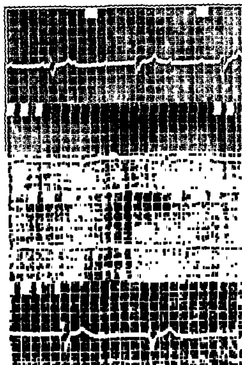


Fig 29—Complete heart block. (Courtesy Dr H K Mohler)

where along its pathway and causes delayed, partial, or complete heart block.

Etiology Heart block is generally acquired, rarely congenital. Acquired heart block may be caused by injury of the auricular musculature, in the A-V node or in the bundle of His, in the pathway between the sinoauricular node and the auricle, in the bundle branches, in the ventricular myocardium, or in the arborization of the Purkinje fibers. Heart block may be brought about by syphilis, arteriosclerosis, rheumatic fever, and

other febrile diseases; by coronary disease, emboli, toxic agents, and other conditions that may cause severe myocardial damage; also by digitalis, strophanthus, aconite, physostigmine, morphine, nicotine, and potassium salts.

Types of Heart Block 1 Complete Heart Block (auriculoventricular block) The auricles and ventricles each have their own rhythm The ventricular impulse arises within the ventricle and is independent of the auricle The heart rate is slow, from 30 to 40 per minute, and, occasionally, the ventricular rate may be as slow as 8 to 10 per minute and is accompanied by attacks of Stokes-Adams' syndrome (giddiness, faintness, unconsciousness, muscular twitchings, or convulsions). The auricular rate is fast

Graphically, complete heart block is recognized by the extremely slow ventricular rate, 30 to 40 per minute, while the auricular wave is rapid The Q-R-S complex is often distorted, presenting notching of the limbs or apex and at times distinct arrhythmia The P waves (auricular) are rapid, regular and have no relation to the Q-R-S complex, though at times they are notched Deformity of the waves may at times occur as the result of the P wave superimposing upon the R and Q waves Partial heart block presents more rapid ventricular beats than complete heart block though dissociation of P and Q-R-S waves is noted

2. *Partial Block*· When the block is incomplete, the heart rate is faster than in the complete block, indicating that some of the auricular beats come through to the ventricle.

3. *Sinoauricular block* causes a drop in the rate of both the auricle and ventricle. The heart rate is slow and the pause is lengthened. This may be brought on by large doses of digitalis and vagal pressure. It may be abolished by increasing the heart rate by atropine, deep breathing, exercise, or swallowing

4. *Dissociation by interference* is due to myocardial degeneration and occurs when a new impulse arises before the heart has sufficient time to recover from

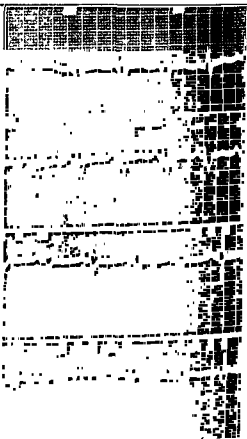


Fig 30—Right bundle branch block (Courtesy Dr. H. K. Mohler)

the previous impulse This is seen in auricular fibrillation, auricular flutter, and ventricular extrasystole In this condition the A-V node is affected

5 *Intraventricular block* is of three types.

(*a*) *Bundle Branch Block:* Either the right branch of the bundle of His or the *left branch* of the bundle of His may block the impulse from entering the

right ventricle or the left ventricle These abnormalities are discernible on the electrocardiogram

Right Bundle Branch Block (block of right main branch of the auriculoventricular bundle): The distortions occur in the ventricular complexes Each ventricular complex is preceded by a normal P wave in all leads The ventricular complexes show a widening of the Q-R-S complex exceeding 0 1 of a second in all leads. The S wave in lead I descends quite low and the R wave in lead III extends upward quite high. The T waves point upward in lead I and downward in lead III. That is, in opposite directions to the S and R waves in the first and third leads. The T wave in lead II may point in any direction (This was formerly considered as left bundle branch block)

Left Bundle Branch Block (block of left main branch of the auriculoventricular bundle): Each ventricular complex is preceded by a normal P wave. The Q-R-S complex is widened, exceeding 0.1 of a second in all leads. The R wave in lead I ascends high, the T wave points downward in this lead In lead III, the S wave descends quite low and the T wave points upward; that is, in the opposite directions of main initial deflections in leads I and III. (This was formerly considered as right bundle branch block.)

To differentiate between left and right ventricular preponderance (SEE: Figs 40 and 41, pp 437-8) and left and right bundle branch block, it should be noted that in bundle branch block the R wave is wider than normal and is usually notched or splintered and the T points in the opposite direction to the S and R; while in ventricular preponderance the Q-R-S complexes are not widened and the T points in the same direction as the main initial defections in leads I and III.

(*b*) *Arborization block* occurs when there is an interference with the conduc-

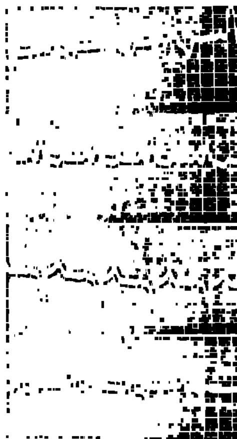


Fig 31—Left bundle branch block (Courtesy Dr H. K. Mohler)

tion through the terminal division of the Purkinje fibers (subendocardial fibers)

(*c*) *Advanced intraventricular block* (diffuse type) may give rise to gallop rhythm in which there may occur doubling of the first or second heart sounds

The various types of heart block may best be diagnosed by the electrocardiogram.

of the prematurity of this beat, the P wave of that contraction is invisible, or it may immediately precede the premature ventricular contraction Ventricular premature contractions of right ventricular origin are identified in lead II, by the upward directed distorted R wave, while that of left ventricular origin, shows a downward distorted R wave, which assumes an upward direction in lead II.

The Interpolated Extrasystole In some instances after the ventricular extrasystole, there is a normal response to the normal auricular systole, causing a ventricular beat which can be appreciated in the radial artery without a corresponding auricular beat being discernible in the jugular.

Auricular Extrasystole. The heart sounds and radial pulse are identical with the ventricular extrasystole Only by pulse tracing and electrocardiogram (jugular and radial) can this condition be recognized.

Extrasystole Arising in the Auriculo-ventricular Node (nodal extrasystole, Mackenzie): In this class there is a simultaneous premature contraction of the auricles and ventricles The condition may be ascertained only by arterial and venous pulse tracings and by the electrocardiogram All forms of arrhythmia may be distinctly classified by the electrocardiographic tracings

Simultaneous Occurrence of the Normal Auricular Systole and of the Ventricular Extrasystole In these cases the heart's action is rather slow. The auricles and ventricles contract simultaneously, so that the auricle is prevented from emptying its contents into the ventricle, thus sending a large wave into the jugular, and at the same time causing an absence of the radial pulse.

3. ***Auricular Fibrillation:*** This type of irregularity is the one most frequently encountered. It is characterized by a complete disorganization of rate, regularity and force The irregularity is at its maximum when the heart rate exceeds 120 per minute; when the rate is slowed to about 80 per minute the irregularity is less prominent When listening to the cardiac apex, the heart sounds are heard as a medley of sounds varying in intensity, rate, rhythm and quality. No two sounds are alike; there are a number of tumultuous sounds in rapid succession, then there may be several loud isolated sounds interspersed with comparatively long pauses; this may be followed by one or by several either normal heart sounds or rudimentary sounds. The irregular irregularity of the heart's actions are the distinguishing features of auricular fibrillation. The pulse rate here does not keep pace with the heart rate; many of the rudimentary cardiac impulses do not reach the wrist, therefore there is a pulse deficit. A heart rate of 120 may present a pulse rate of only 100 or less Thomas Lewis describes the pulse of auricular fibrillation as follows· "The pulse is a medley of beats of many sizes, an intimate mingling of changing pauses; now the beats are almost uniform in strength and spacing, now feeble pulsations chase along rapidly; now the pulse is lost; now it returns with increased vigor" The sphygmomanometric reading is quite characteristic. A few isolated systolic heart sounds may be heard over the cubital fossa when the cuff is compressed at 160 mm., several more at 150 mm; at 130 mm many more beats are transmitted These are of varied strengths. Near the beginning of the diastolic phase most of the beats, strong and

weak, regular and irregular, are heard with ease. The point where most beats are first heard may be designated as the systolic pressure of the individual.

When the heart rate is slow it is often difficult to diagnose auricular fibrillation; many of the rudimentary beats do not occur; the wild delirium of the heart is not as evident as when the rate is fast, nor is the pulse deficit as marked The irregular spacing and the occasional disturbance in force and rhythm of the beats discloses the type of irregularity. Occasionally slow auricular fibrillation may resemble extrasystole. To differentiate these conditions the heart rate is sped up by exercise, strychnia or atropine. If, when the heart rate becomes faster, the irregularity becomes more pronounced, the condition is most likely auricular fibrillation. On the other hand, when the heart is slowed by rest or digitalis and the irregularity becomes more evident, then the condition is usually extrasystole.

Auricular fibrillation occurs in severe myocardial degeneration of either the ventricles or the auricles The irregularity may be transient or permanent In acute infections, in thyrotoxicosis and in other infections in the young it may be a temporary derangement In arteriosclerosis, in severe myocarditis, in coronary infarction, in severe heart damage following rheumatic disease and in the myocardial degeneration of the aged, the irregularity is permanent and is accompanied by other signs of cardiac decompensation.

Prognosis Auricular fibrillation resulting from mitral stenosis is, with moderate care, compatible with long life Two such patients under my care have been fibrillating steadily for 30 years, though during that period both have had several attacks of heart failure from which they recovered. In thyrotoxicosis the irregularity usually disappears after thyroidectomy or when the thyrotoxic manifestations are otherwise controlled. The irregularity occurring during infectious diseases often disappears after complete convalescence In the aged, in arteriosclerosis, in severe myocarditis and following coronary infarction, particularly when there are other signs of gross cardiac decompensation, the prognosis is poor and the span of life is materially shortened; severe cases seldom survive two years. Auricular fibrillation is rare in syphilitic myocarditis When this irregularity accompanies aortic regurgitation, embolic phenomena are of frequent occurrence. The presystolic murmur of mitral stenosis in cases of auricular fibrillation may become inaudible or may appear as a systolic murmur or its timing may become extremely difficult during periods of cardiac decompensation. An early sign of return of cardiac compensation is the return of the murmur.

Mechanism of Auricular Fibrillation Auricular fibrillation is the result of an abnormal impulse traveling an abnormal course. According to Lewis the cardiac impulse or "the wave circulates continuously over the auricle at the rate of about 450 per minute The movement is irregular in that the same path is not followed precisely from cycle to cycle." This rapid movement instead of causing full contraction of the auricles produces contractions of only individual muscle fibers. These impulses are transmitted to the ventricles at irregular times with varying force and are partially blocked in their passage through the auriculo-ventricular bundle or its branches. The

The arteries are studied as to their tension, the amount of visible pulsation, and the condition of the pulse. The radial artery is the most frequently studied in order to estimate the force of the cardiovascular system. Other arteries should also be studied by inspection, palpation, and at times by auscultation.

Inspection: For a thorough inspection of the entire superficial arterial tree, the patient should sit or lie, with his arms elevated, *so that his hands rest upon his head*; when in this position, the axillary, brachial, radial and other arteries, when pulsating, can readily be detected. Visible pulsation in all the superficial arteries is usually *an indication of aortic regurgitation*, it may also be noted after exertion, in the presence of arteriosclerosis, in exophthalmic goiter and in certain anemias Local arterial pulsation may be caused by *partial compression of the* main artery supplying that part. Visible pulsation in the neck and the arms alone may be due to aneurysm of the arch of the aorta, arteriosclerosis, or tricuspid *regurgitation.*

Palpation: Besides studying the pulse and determining its character, palpation is employed to differentiate a pulsating artery from a pulsating vein, particularly if the pulsation is in the neck.

To differentiate arterial from venous pulsation, the index finger should be placed midway between the clavicle and the angle of the jaw, directly upon the pulsating vessel If the pulsation is stopped at the point of compression, so that pulsation is noticed below the point of compression and none above it, it is an indication of arterial pulsation. But, if the pulsation is intercepted from above downward and the vessel is seen to be filling from above downward, it is an indication of venous pulsation.

Percussion: *Percussion in the examination* of an artery is employed only for the sake of determining the possible area of dullness caused by aneurysm

Auscultation: Normally, *no sound is elicited over a pulsating artery unless* that artery is partially compressed. A "pistol-shot sound" is heard in the femoral arteries in cases of aortic insufficiency, and at times also in hypertension *Duroziez's sign* is a peculiar murmurous to-and-fro sound heard over the femoral, carotid and subclavian arteries in cases of aortic regurgitation, when the arteries are slightly compressed. A very loud *systolic murmur may at times* be heard at the aortic orifice, and when it is accompanied by an accentuation of the second aortic sound it is indicative of aortitis.

A soft systolic murmur, because of fatty degeneration, hypoplasia, or any other chronic disease of the arteries, is often heard over the innominate and carotid arteries when the vessels are markedly relaxed A functional systolic murmur is sometimes heard in these vessels in cases of anemia. A systolic *murmur may at times be heard over the* intercostals in coarctation of the aorta

A loud, systolic, "whiffing" *sound is* heard over the subclavian artery (below the clavicle) at the height of inspiration *This murmur is attributed to pleural adhesions or to some other intrathoracic* condition which apparently compresses the artery during inspiration It is frequently met with in apical pulmonary tuberculosis. Aneurysms of the subclavian artery is characterized by expansile pulsation, thrill and bruit, *difficulty in deglutition, and, at times,* hoarseness.

Disease of the Arteries

Arteriosclerosis

Arteriosclerosis (Gull-Sutton's disease) is a chronic disease of the arterial system characterized by degeneration of the arterial walls accompanied by infiltration with fibrous tissue and lime salts, causing thickening and loss of elasticity of the vessels with narrowing of their lumen. The disease may be diffuse or circumscribed.

The diffuse type of sclerosis may affect: (1) The entire arterial tree (*arteriosclerosis*); (2) the capillaries (*arteriocapillary fibrosis*), (3) the veins (*phlebosclerosis*), (4) the entire vascular system (*angiosclerosis*).

Circumscribed arteriosclerosis may affect part of one or more arteries (*atheroma*)

Atherosclerosis is a type of arteriosclerosis in which there is atheromatous degeneration of the connective tissue of the arterial walls

Mönckeberg's sclerosis is a primary degeneration of the media in the large and medium-sized muscular arteries of the periphery. The lumina of the affected arteries become wider than normal

Etiology: Arteriosclerosis is a physiologic process in old age After the fiftieth year the arteries usually harden, lengthen, become more tortuous and their caliber diminishes Hereditary influence may cause hypertension in young individuals, the cause of which is otherwise unexplainable Pathologically, arteriosclerosis may be brought about by syphilis, alcoholism, worry, stress and strain, overwork, overeating, intoxications by lead and arsenic, intestinal toxemia, focal infection and sympathetic nervous disturbances Disease of the kidney may cause arteriosclerosis or may be caused by it.

General Symptoms: 1. Hypertension is usually associated with most forms of arteriosclerosis, though in the senile who present hard, pipestem arteries, the pressure is often abnormally low. Hypertension is found in three groups.

(*a*) Simple hypertension without apparent renal or cardiac disease (hyperpiesia). This may be the result of angioneurosis, or an early stage of arteriosclerosis before external signs are manifested Essential hypertension is a distinct entity; its cause is as yet unknown (SEE: p 412).

(*b*) Hypertension due to manifest arteriosclerosis.

(*c*) Hypertension associated with renal or cardiovascular-renal disease.

2. The superficial arteries are hard to the touch and tortuous.

3. Pallor, digestive disturbances, fatigue on moderate exertion, rapid aging, polyuria, and, in men, enlarged prostate

Local Symptoms: ***Heart:*** Myocarditis with cardiac hypertrophy and accentuation of the second aortic sound, and occasionally angina pectoris occur. Cardiac hypertrophy may be followed by dilatation and decompensation.

Lungs: There may be chronic bronchitis and emphysema

Eyes: The retinal vessels are tortuous and sclerotic.

Brain: There may be dizziness and signs of cerebral anemia, hemorrhage or thrombosis

Kidney: Arteriosclerotic kidney is characterized by polyuria of low specific gravity containing little albumin, few casts and may or may not be associated with nitrogen retention in the blood

Vasomotor Symptoms: Sensation of fullness or lightness in the head, coldness

The arteries are studied as to their tension, the amount of visible pulsation, and the condition of the pulse. The radial artery is the most frequently studied in order to estimate the force of the cardiovascular system. Other arteries should also be studied by inspection, palpation, and at times by auscultation.

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may be narrow throughout its length or constricted at a certain level as in coarctation.

Anomalies in Structure: The aorta may structurally resemble that found

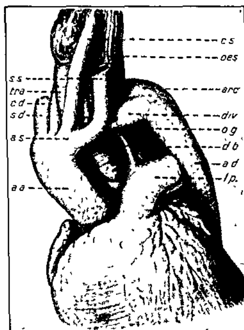


Fig 2—Drawing of congenital defect of aorta The right-sided aorta passes over the right bronchus, then behind the trachea and esophagus. The ring around the trachea and esophagus formed by the right-sided aorta and the left aortic arch (left subclavian artery, occluded short vessel, and diverticulum) is distinctly visible

ss left subclavian
tra trachea
c.d right carotid
a s. left innominate
a a. ascending aorta
c.s left carotid
oes. esophagus
arc. arch of aorta
div diverticulum
o g. occluded vessel
d b. ductus Botalli
a d. descending aorta
l p left pulmonary artery

(Courtesy, Dr. Aaron Arkin, American Heart Journal)

normally in some quadrupeds, reptiles or birds In the *quadruped* type the aorta divides into an ascending and a descending trunk. The ascending trunk is directed vertically upwards and subdivides into three branches to supply the head and upper extremities. In the *reptilian* type the aorta divides near its origin into two branches which, after a short run, reunite The esophagus and trachea pass between the two branches In the *avian type* of aorta the arch passes over the right main bronchus and continues on the right side or it may be behind the esophagus and trachea

There may also be *absence of the aortic arch.* The arch of the aorta may be entirely absent, or only the isthmus (portion lying between the origin of the subclavian artery and the insertion of the ductus Botalli) may be closed or entirely absent. This shuts off the communication between the ascending and descending aorta. The ascending aorta then supplies the vessels of the head and the right subclavian, and the open ductus Botalli goes over into the descending aorta on the left side.

Aaron Arkin* reported and described six cases of *"Double aortic arch with total persistence of the right and isthmus stenosis of the left arch."* This type of lesion represents an intermediate type between persistence of both aortic arches and persistence of the right aortic arch In his cases there were persistence of both aortic arches. Because Arkin was able to demonstrate roentgenologically the presence of right-sided pharyngeal aorta and the left dorsal aortic root which looks like a diverticulum and lies behind the esophagus, he named this anomaly "Right-sided esophageal aorta or total persistence of the right and pharyngeal stenosis of the left aortic arch."

*Arkin, Aaron· Am Heart J, 11: 444 (April) 1936

and blanching of the extremities, numbness and tingling sensation in the hands and feet often accompanied by congestion or cyanosis

The Lower Extremities: The symptoms engendered by arteriosclerosis of the lower extremities are similar in many respects to those caused by other forms of peripheral vascular disease (SEE: p. 535).

Aortitis

When the aorta is affected by sclerotic changes, an atheromatous plaque may

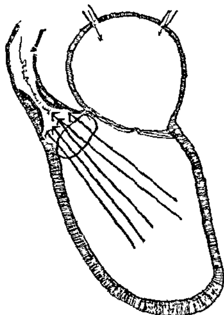


Fig. 1—Aortitis.

be formed in the intima, or other degenerative changes may become manifested. These may lead to acute or chronic aortitis and eventually to aneurysm. Syphilis is the greatest single predisposing factor to this condition. Other factors are arteriosclerosis and rheumatism

Aortitis usually affects the first portion of the aortic arch near the coronary orifices. In chronic aortitis, the greater portion of the aorta may be affected.

Symptoms: The symptoms are pain in the upper sternal region, or a sense of fullness on exertion, the pain frequently radiating to the arms. The pain often comes on when the patient is in bed and is relieved on getting out of bed and assuming an upright posture, or leaning somewhat forward supported by the hands. Dyspnea and a sense of precordial oppression resembling angina pectoris are often experienced. Occasionally there are no chest symptoms.

Physical Signs: ***Inspection:*** Pulsations in the vessels of the neck and suprasternal notch.

Percussion: Increased area of sternal vascular dullness to the right and left of sternum above third rib

Auscultation: Accentuated aortic second sound; at times also a harsh systolic murmur over the second right intercostal space (aortic area) transmitted into the right carotid. This is to be differentiated from aortic stenosis chiefly by the presence of an accentuated second aortic sound. In aortic stenosis the second sound is very weak or absent.

Congenital Anomalies of the Aorta

The aorta may show anomalies in position, size, structure and origin of its arteries.

Anomalies in Position: Normally the aortic arch reaches to about 2.5 cm below the suprasternal notch Occasionally it may reach to the top of the sternum, or may be displaced downwards to as low as 4 to 8 cm. below that point. It may arch over the root of the right lung instead of the left and finally descend along the left side of the spinal column This is usually associated with situs inversus viscerum.

Anomalies in Size: The aorta may be of larger than normal caliber or it

The following are seven of the clinical signs upon which Arkin based his diagnosis:

"1. Dullness on percussion along the right sternal border upward to the head of the right clavicle.

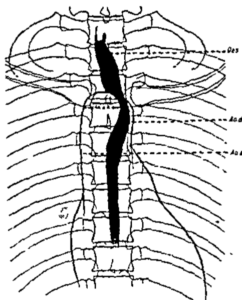


Fig. 5—Drawing of x-ray plate of right- [illegible]

appears prominent

Ao. a b., width of ascending aorta
Ao d, descending aorta.
Ao a, ascending aorta.
Oes, esophagus

(Courtesy, Dr. Aaron Arkin)

"2. Visible systolic pulsation in the second or third right intercostal space near the sternum, or in the right supraclavicular fossa.

"3 Palpable strong pulsation in right supraclavicular fossa.

"4 Maximum intensity of the aortic heart sounds to the right and above the *usual location* (Often in the right supraclavicular fossa)

"5. Displacement of the trachea slightly to the left.

"6. Tracheal tug.

"7. Delay in the passage of a rigid stomach tube at the level of the third dorsal vertebra with pulsation transmitted along the tube."

The x-ray findings described by him were as follows:

"(1) A shadow to the right of the sternum, running upward to the head of the right clavicle, with a distinct systolic pulsation; (2) slight displacement of the trachea, and definite displacement of the esophagus to the left; (3) absence of the normal aortic knob on the left side, or only a small shadow of the descending arch on the left side; in some cases two aortic knobs, one on each side; (4) in the right oblique position the aortic knob lies behind the trachea and esophagus, both of which are displaced forward and to the left (most characteristic of all is the circular forward displacement of the esophagus by the arch of the aorta); (5) shadow of the diverticulum either in the retroesophageal knob or in the shadow of the descending arch on the left side; (6) in the left oblique position a wide shadow of the ascending aorta to the right of the trachea and evidence that the aortic arch runs behind the esophagus to reach the left side."

For ***Coarctation of the Aorta*** and ***Congenital Aortic Stenosis***, SEE: p 508.

Aneurysm

An aneurysm is a localized expansion or dilatation of the lumen of an artery It is usually circumscribed in shape.

Aneurysms are classified as: I. *False*. II. *True*

I. **False Aneurysm:** This term is applied to a circumscribed collection of blood outside the vessel due to rupture

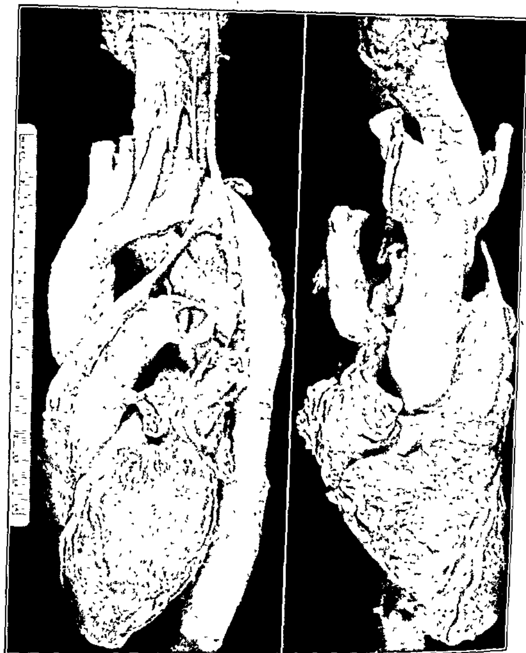


Fig. 3—Photograph of specimen of congenital malformation of the aorta, showing the double aortic arch. The right arch lies behind the trachea and esophagus, and the left arch (left innominate and subclavian artery, isthmus stenosis, and left dorsal root) in front The ductus arteriosus communicates with the left dorsal root. (Courtesy, Dr. Aaron Arkin)

Fig. 4—Same as Fig 3 viewed from the right side showing the aortic arch behind the trachea and esophagus, with the left arch in front (Courtesy, Dr. Aaron Arkin)

Palpation: If the enlargement is superficial and not too thoroughly organized, expansile pulsation and a thrill may be palpated in the second and third interspaces to the right or to the left of the sternum.

Percussion will reveal an increased area of dullness over the manubrium.



Fig. 7—Aneurysm of the aortic arch.

Auscultation may at times reveal a *bruit* occurring during both the systole and diastole in the second interspace, either to the right of the sternum or near its left border. A *bruit* is not heard if the aneurysmal clot is large and the expansion not well marked.

Aneurysm of Transverse Portion of the Aortic Arch: Symptoms are: Dyspnea; dry brassy cough, caused by pressure on the trachea; dysphagia when pressure is exerted on the esophagus; bronchitis, when pressure is exerted over a bronchus; brassy cough and aphonia from pressure of the left recurrent laryngeal; pupillary changes from pressure on the upper dorsal and the lower cervical ganglia; rapid emaciation, when the aneurysm presses on the thoracic duct.

Physical Signs: When the aneurysm is large enough, ***inspection will*** show a tumor in the middle line or to the right of the sternum

Tracheal tugging may be elicited by *palpation*, particularly when the aneurysm is in close proximity to the trachea or the larynx. Inequality of both pulses

of the artery; in other words, the sac ot the aneurysm is *partly or entirely* formed by surrounding tissue or by a newly-formed fibrous covering.

II. **True Aneurysm:** This is a more or less localized dilatation of an artery. The aneurysmal sac is composed of the layers of the arterial wall. The dilatation may be fusiform, saccular or cylindrical. A *dissecting aneurysm*, which belongs to the "true aneurysm" type, is one in which the intima has ruptured and the blood forced itself between the layers of the arterial wall some nerve. The signs common to all *aneurysms which* are not organized and are superficial, are expansile pulsation and *bruit*.

Aneurysm of the Aorta: This condition most frequently occurs in the ascending portion of the arch and gives rise to many phenomena. The next commonest site is the transverse portion; third, the descending portion of the arch of the aorta. The male sex, middle life, laborious work, syphilis, rheumatism, *gout and alcoholism are predisposing* factors. In other words, any factor that leads to arterial degeneration on the one hand and to abnormally great vascular tension on the other may produce aneurysm.

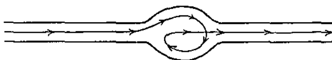


Fig. 6—Aneurysmal dilataton of an artery.

Physical Signs: The cardinal physical signs are applicable only to superficial aneurysm *Inspection* shows bulging, or a pulsating tumor, if the aneurysm is not covered by bone (ribs or sternum). *Palpation* will disclose an expansile pulsation and a thrill. *Percussion* elicits circumscribed dullness. *Auscultation* discloses a *bruit.*

Etiology: The *commonest cause* for aneurysm is a weak point in the walls of an artery, usually due to syphilis. Aneurysm may also occur in nonsyphilitics, as the result of sudden strain or an injury.

Symptoms: The symptoms of aneurysm depend entirely upon the location, the size, and the amount of pressure it exerts upon its adjacent structures Pain, however, is the most constant symptom of aneurysm, particularly in the very early stages, when the intima is being stretched or ruptured. After an aneurysm has attained a considerable size, pain may be produced by pressing upon

Aneurysm of Ascending Portion of the Aortic Arch: ***Symptoms:*** When *the aneurysm* is large and presses against the recurrent laryngeal nerve, aphonia, dyspnea and brassy cough are prominent *symptoms.*

Physical Signs: *Inspection* shows a tumor to the right, rarely to the left, of the sternum over *the second* and third interspaces. The veins of the neck, head and upper extremities may be distended when the aneurysm *is large* enough to cause pressure upon the superior vena cava. When the pressure is exerted upon the subclavian artery, edema of the right arm is noted. When the aneurysm is sufficiently large to cause pressure on the inferior vena cava, swelling of the lower extremities will be noted. The apex beat is usually not placed to any extent towards *the left*

may reveal expansile pulsation. *Percussion* elicits dullness. *Auscultation* reveals a *bruit,* heard directly over the tumor mass The most accurate diagnosis of aneurysm in this region is made by means of the fluoroscope and roentgenogram

Aneurysm of the Innominate Artery: The innominate artery may be involved independently or in association with aneurysm of the aorta

Symptoms: The principal symptoms of aneurysm of the innominate artery are throbbing and pain at the root of the neck, dysphagia, dyspnea and at times, stertorous breathing

Physical Signs: Inspection reveals pulsations in the right supraclavicular region, with bulging or dislocation of the right sternoclavicular joint On *palpation* it is found that the right radial pulse is retarded and more compressible than the left; the right external jugular vein is usually distended, and is accompanied by right-sided edema of the face and neck. Tracheal tugging is often elicited, as is also expansile pulsation and a diastolic shock over the site of the tumor. *Percussion* elicits dullness over the right sternoclavicular region, and upon *auscultation* a *bruit* may be heard in the right supraclavicular region, and often also in the first interspace close to the sternum

Aneurysm of One of the Auricles or Ventricles: When this occurs, the diagnosis is usually made by the x-rays

Arteriovenous Aneurysm: This is caused by an abnormal communication between an artery and a vein When the communication is direct it is known as "aneurysmal varix"; where the sac intervenes between the artery and the vein it is termed "varicose aneurysm." Arteriovenous aneurysm is often met with in the peripheral vessels, and is usually the result of some form of traumatism or of syphilis. It may be seen in the popliteal space, in the groin, in the axilla, in the subclavian, and in the bend of the elbow.

Differential Table of Aortic Aneurysm

Inspection

Ascending Portion	Transverse	Descending	Abdominal Aorta
Bulging of the thorax and pulsating tumor are present to the right of the sternum, in the second and third intercostal spaces, except when the aneurysm projects upward and inward from the lesser curvatures.	Bulging and pulsation in the episternal notch.	Bulging and pulsation to the left of the sternum, usually in the second and third left interspaces, near the sternum, or very rarely, in the left scapular regions	Pulsating tumor in the abdomen frequently causing expansile pulsation over a limited area
Apex of heart is usually displaced downward and outward.	Apex generally in the normal position.	Apex beat is displaced to the right and two areas of pulsation are seen.	Apex beat not displaced

If pulsation is synchronous with the systole of the heart, and erosion of the chest wall has occurred, there will be a more or less prominent pulsating tumor over which the skin is livid or necrotic and it may be the seat of hemorrhagic oozing

occurs when the innominate, the left carotid and the subclavian arteries are involved. If the sternum has been eroded, an expansile pulsating mass may be palpated over the upper part of the sternum and a little to the right of it. *Percussion*

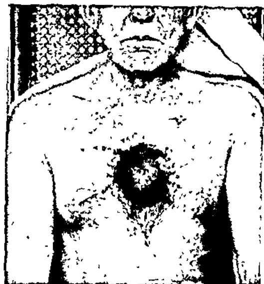


Fig. 8—Aneurysm of thoracic aorta, with erosion of the sternum

will reveal dullness to the right or to the left of the sternum, as the case may be. *Auscultation*, when the aneurysm has not become entirely organized, will reveal a *bruit* audible over the upper part of the sternum

Aneurysm of Descending Portion of the Aortic Arch: This form of aneurysm may exert sufficient pressure against the vertebrae to cause erosion and destruction

Symptoms are extreme pain and often angina pectoris and occasionally hiccoughs. Swelling may appear posteriorly in the scapular region or in the left infraäxillary region; dysphagia often results from pressure upon the esophagus; bronchiectasis, abscess or gangrene of the lung may be produced by pressure of the aneurysm against a bronchus.

Physical Signs: *Inspection* is of no value before the appearance of a tumor mass posteriorly. When the tumor mass does appear, a pulsation may be seen over the mass. Expansile *pulsation* may be felt over the tumor and fluctuation may be elicited if the aneurysm is not thoroughly organized. *Percussion* elicits dullness over the affected part. *Auscultation* reveals pulsation or a *bruit* heard posteriorly in the vicinity of the fifth or sixth dorsal spine.

Aneurysm of the Descending Thoracic Aorta: ***Symptoms*** are pain and, because of partial compression of the lung, dyspnea. A mass may appear upon the lower thorax to the left or right of the spinal column.



Fig. 9—Aneurysm of thoracic aorta, with erosion of the seventh and eighth ribs pointing posteriorly

Physical Signs: On *inspection* a bulging or a tumor mass may be noted when the aneurysm is large. *Palpation* shows that the tumor mass is usually soft and apparently compressible and

ngling and numbness of the affected part when held in certain postures. Pain when in motion, or intermittent claudication in advanced cases.	Tingling, numbness and pain in various parts of the foot and leg.
antar ischemia usually associated with obliterated dorsalis pedis pulsation	Plantar ischemia may occur in the presence of palpably pulsating dorsalis pedis and other arteries.
e disease is slowly progressive and has a tendency to develop a collateral circulation	No tendency to form a collateral circulation.
elling, redness and pain in affected foot when in the dependent position. One leg may be affected at first.	Generally no swelling, the skin feels dry, scaly and may be fissured; generally a bilateral affection from the start.
ldness and cyanosis.	Coldness and pallor.
efore gangrene sets in, the toenails may not be affected.	Dry, brittle and discolored toenails
-ray examination will not show calcareous vessels.	X-ray examination of the extremities may show generalized calcareous infiltration of the arteries.

angrene may be caused by thrombo-itis obliterans, by arteriosclerosis, etes, Raynaud's disease and embolic ases

Raynaud's Disease

he etiology of Raynaud's disease is nown It appears to be a peripheral ospastic disease affecting all the four remities, the tip of the nose and occa-ally other acral parts. This disease more prevalent among women than n. In the milder forms, exposure to d, reaction to excitement or to pain cause blanching of the fingers and s accompanied by numbness and a gling sensation This blanching is fol-red by redness or cyanosis with a sation of heat. These attacks may several minutes to an hour; they y be relieved by friction of the parts immersion in warm water. In severe es there may be localized small trophic erations of the skin, scleroderma, or phic changes in the fingernails and nails. Arterial pulsations remain rmal.

Erythromelalgia

This is caused by excessive localized sodilation of both feet, though one foot alone and occasionally an upper extremity may be affected. The etiology is unknown; it may occur in either sex. The outstanding symptoms are redness and intense burning pain in the affected part when kept in the pendent position These attacks come on at irregular intervals and may be relieved when the affected part is elevated or immersed in cold water. During the attack the part is red and hot and the superficial vessels are distended and pulsate (SEE: p 885).

Essential Thrombophilia

This is a type of thrombosis usually occurring in the medium sized arteries without any demonstrable arteriosclerosis or inflammatory changes in the arterial walls. The symptoms depend upon the site of the lesion There may be pain and occlusive symptoms in the parts supplied by the cerebral, retinal and visceral arteries as well as by the arteries supplying the extremities The coagulation time of the blood is usually diminished. The etiology is unknown; such cases were observed after electric shock, burns and after trauma.

arteriosclerosis obliterans, Raynaud's disease, erythromelalgia, essential thrombophilia and periarteritis nodosa.

Symptoms common to peripheral vascular disease, irrespective of cause, are pain, numbness and altered circulation.

Pain is the most outstanding complaint; it varies in intensity, character and distribution depending upon the site affected and the severity of the disease. In the early stages of lower extremity affection, when the occlusion is limited to the digital or plantar vessels there may, after walking only a short distance, be either a burning sensation or a sharp pain in the foot which may radiate to the calf muscles Numbness in one or more toes or in the foot may accompany the pain or it may occur independent of pain; numbness may occur during exercise or when at rest Numbness of the finger tips is an early manifestation of occlusion or spasticity of the peripheral vessels of the upper extremities Intermittent claudication occurs in late stages of vascular occlusion The pain in the calf of the legs is brought out by walking and is described as a severe cramp. It usually stops when resting Pain in the buttock, after walking, which radiates downwards may be caused by spasm or partial occlusion of the inferior gluteal artery. As the occlusive disease progresses the pain becomes aggravated and may be continuous, even when at rest. Coldness, numbness and cessation of perspiration in the affected parts may precede pain or may accompany it. Blanching of the affected part may accompany numbness and precede pain Hyperemia of a part and often deep cyanosis may occur with pain.

Thromboangiitis Obliterans (Buerger's Disease)

This is a disease of the blood vessels occurring in young or early middle-aged men, causing occlusion thrombosis in the arteries and phlebitis in the veins. In this disease the veins as well as the arteries are affected, thus differing from arteriosclerosis obliterans, in which the arteries alone are affected This disease is characterized by excruciating pain in the foot, leg or arm, usually worse during the night The extremity affected is cyanotic, cold and clammy. When the affected part is lowered it rapidly becomes congested but blanches just as rapidly when elevated Pulsation in the dorsalis pedis, posterior tibial or the arteries of any affected part is either decreased or obliterated. Heat and cold sense is diminished; pain is a prominent symptom, and gangrene may occur in the toes, foot or in any other parts affected by thromboangiitis obliterans.

Differential Table

Thromboangiitis Obliterans* versus *Arteriosclerosis Obliterans

Thromboangiitis Obliterans	Arteriosclerosis Obliterans
Affects the arteries and veins.	Affects the arteries exclusively
Migrating phlebitis common.	No migrating phlebitis.
Possibly of inflammatory nature, most prevalent among males	Metabolic in nature, structural changes in the intima, noninflammatory; occurs in both sexes.
May be a familial predisposition.	Not usually a familial predisposition.
Occurs principally in young and early middle-aged men, or between the ages of 15 and 45.	Most prevalent past middle age.

Prognosis: The disease may run from a few weeks to a few months Recovery is rare.

Acute Arteritis

The arteries are resistant to infectious processes, though occasionally acute endarteritis may develop during the course of typhoid fever, septicemia and pneumonia. Localized inflammation of an artery may result from local suppuration, syphilis, tuberculosis, rheumatic fever, or from some infection of the lymphatics or the vasovasorum. Severe infection may cause necrosis and rupture of the vessel with hemorrhage.

Examination of the Capillaries

Despite the fact that they are the smallest of the blood vessels, the capillaries, because of their distribution throughout the skin and the other superficial parts of the body, are of great importance in the circulatory system What is usually termed "the complexion" of an individual depends largely upon the degree of fullness or emptiness of the capillaries in the skin. Thus, a flushed skin means full capillaries, and *per contra*, pallor means comparatively empty capillaries

Capillary pulsation is a prominent symptom in aortic regurgitation (Quincke's capillary pulse). This pulsation consists of a periodical waxing and waning of the skin color, synchronous with the apex beat and the carotid impulse It is observed upon the fingernails, lips, and upon the forehead, when the skin is briskly rubbed In order to bring out this pulsation more prominently in the fingernails, the patient's hand is supported, and a finger held lightly between the examiner's thumb and forefinger below the first metacarpal joint. Very gentle pressure is then brought to bear on the lateral surfaces of the finger. If capillary pulsation be present, it will thus be noted readily at the roots of the nails. Gentle pressure upon the fingernail will often accentuate the capillary pulsation in the nail. A flashlight held underneath the fleshy part of a distal phalanx will reveal a pulsation beneath the fingernail. This pulsation, when



Fig 12—Inspecting lip through glass slide for capillary pulsation in case of aortic regurgitation

present on the lips, can be brought out more clearly by pressing a glass slide upon the mucous membrane of the lower lip Capillary pulsation, while always a prominent sign in aortic regurgitation, is at times also observed in exophthalmic goiter and in certain anemias, particularly when associated with disease of the peripheral arteries Cases have also been reported where capillary pulsations occurred in otherwise healthy persons after fatigue.

Examination of the Veins

Only superficial veins lend themselves to physical examination. They are examined chiefly by ***inspection*** and ***palpation.***

Embolic Occlusions of the Arteries

These may result from vegetative endocarditis, from the breaking off of portions of a thrombus and forming emboli as seen in mural thrombosis of coronary origin, in auricular fibrillation and in myocarditis. These may cause localizing signs such as *aphasia, hemiplegia or* sensory disturbances, when the cerebral vessels are affected. When a peripheral vessel becomes occluded there will be sudden pain, blanching and cessation of arterial pulsation below the point of obstruction If occlusion occurs in any of the viscera there may be pain and interference with the function of that part

Periarteritis Nodosa

Periarteritis nodosa is characterized by inflammatory lesions in the smaller and medium sized arteries. All the coats of the arteries are affected, showing hyaline degeneration and inflammation. The

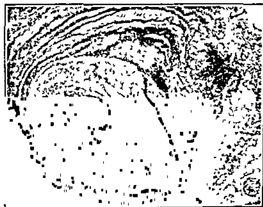


Fig 10—Periarteritis nodosa Photomicrograph of small vessel showing infiltration of the vascular wall, aneurysmal dilatation and thrombosis of the lumen.

lumen of some of the vessels may be thrombotic; others may show aneurysmal dilatations. Small nodules, yellowish white in color, ranging in size from that of a pinhead to a pea, are found on many of the arteries; their number vary from a dozen to several hundred. Occasionally there may be found small nodules on the skin or in the subcutaneous tissue. These nodules are tender or painful to touch.

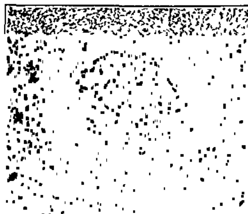


Fig. 11—Periarteritis nodosa Photomicrograph (high magnification) showing the composition of a nodule, the infiltration of the vessel walls and the clotted lumen

The *etiology* is unknown; it occurs more frequently among young males than in females.

Symptoms: The disease may follow a "cold" or any infection; it runs a septic course, with fever, weakness, pain in the muscles, joints and epigastrium There is usually digestive disturbances such as vomiting, diarrhea, melena, and abdominal cramps; occasionally there may be symptoms of mesenteric thrombosis or perforation of the bowel. There may also be cough and hemoptysis, anemia, asthenia and emaciation. The kidneys are affected in over 80 per cent of the cases Hypertension is nearly always present. Leukocytosis and occasionally eosinophilia may be present Painful lesions along the arteries and in the skin, when present, are important diagnostic data.

sure (mediastinal tumor); (*b*) aneurysm of the aorta; (*c*) tricuspid regurgitation; (*d*) severe paroxysms of cough (temporary).

III. **Distention of veins in the arm** may be due to thrombosis or pressure by a tumor, enlarged glands, etc, upon the axillary veins.

IV. **Distention of the veins of either leg** may be due to thrombosis or pressure upon the femoral vein.

V. **Distention of veins of both legs** (varicose veins) may be due to: (*a*) Pressure on the inferior vena cava by abdominal or pelvic tumors; (*b*) ascites; (*c*) thrombosis or pressure upon both femoral veins; (*d*) fecal impaction; (*e*) intrapelvic pressure; (*f*) pregnancy.

VI. **Distention of superficial abdominal veins** may be caused by: (*a*) Compression of the inferior vena cava; (*b*) portal obstruction; (*c*) tumors of the liver; (*d*) atrophic cirrhosis; (*e*) ascites, (*f*) greatly enlarged spleen; (*g*) greatly dilated stomach.

Venous Pulsation

Normally, venous pulsation is not visible in well-nourished individuals; however, in persons who are otherwise normal but are moderately emaciated and have little subcutaneous fat, pulsation in the neck may be readily noted, particularly during respiration. The veins can be seen to fill during expiration and collapse during inspiration, because of negative intrathoracic pressure which at that time draws the blood toward the heart. Swelling of the jugulars during expiration is due to the positive pressure exerted upon the veins, which causes a retrograde wave of blood to close the valve above the jugular bulb.

Pathologically, this pulsation is very much increased in asthma, and chronic emphysema; it is also increased by cough. Adhesive pericarditis usually reverses the filling and emptying of the veins, *i. e.,* the jugular veins fill during inspiration and empty during expiration, because during inspiration the superior vena cava is constricted by adhesions, which hinder the venous flow toward the heart.

Normally, the venous pulse is presystolic in time, or negative, because the veins fill during expiration and empty themselves during inspiration Pathologically, the venous pulse may become systolic in time, or positive, because it may fill during inspiration. It is important, therefore, not to confuse the systolic venous pulse with the carotid pulse. A jugular vein may appear pulsating because of the tranmission from an underlying carotid artery. This can be differentiated by "milking" the vein upward; if the blood does not follow as a venous wave from below, the pulsation is due to carotid transmission. A positive venous pulse due to tricuspid regurgitation usually follows the fingers upward. The normal negative venous pulse can be differentiated from carotid pulsation by compressing the vein near its middle with the finger. Pulsation will cease on the proximal side of the compressed vein, showing that the blood does not regurgitate from the heart. There is also a decided diminution of the undulation on the distal side, which shows that the pulsation is not transmitted from an underlying artery. The presystolic wave of the normal jugular pulse rises slowly, and is followed by a sudden systolic collapse, which in turn is followed by a short interval before the next wave appears. This phenomenon is due to systole of the right auricle, because the right auricle contracts during venous disten-

The veins are inspected for fullness, engorgement and pulsation. Unusual enlargement of the veins is caused by some condition that intercepts the flow of blood to the heart. This obstruction may be general or local.

Diseases of the Veins

General Venous Distention

General venous distention may be caused by:

I. **Failure of the Right Ventricle:** The right ventricle being overfilled, and its walls, having lost their elasticity, cannot propel a sufficient quantity of blood to the lungs for aeration. This produces a certain amount of back pressure, thus causing a general stasis in the venous system. Not only are the superficial veins increased in size but the larger veins, particularly those of the neck, are pulsating, and the surface of the body is cyanosed.

II. **Stasis in the lungs** from any cause, such as emphysema, fibroid phthisis, and pertussis In such cases the lungs are unable to receive all the blood the right ventricle should normally force into them; therefore a certain quantity remains within the right ventricle. This is often the beginning of excessive right intraventricular pressure. When this condition persists, it usually becomes progressive and results in right ventricular dilatation with the symptoms described above.

III. **Compression of the vena cava** by tumors, aneurysm, adhesive pericarditis, or other adhesive bands. These obstructions are purely mechanical; the lumen is constricted and the flow thus intercepted, causing a stasis above the point of compression.

IV. **General convulsion** causes temporary stasis because the muscular contractions, during convulsions, are liable to compress the veins in certain parts of the body.

Local Venous Distention

Local venous distention may be caused either by a tumor or by adhesive bands pressing upon a vein which drains a definite part. Venous thrombosis may have the same effect as does a tumor pressing upon a vein. Disease of the vessel wall may lead to distention.

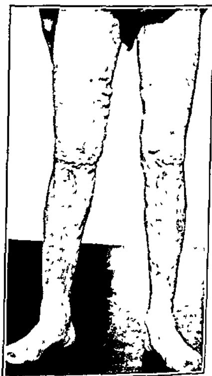


Fig 13—Varicose veins.

I. **Distention of the veins of the scalp** may be due to: (*a*) Tumors of the neck; (*b*) thrombosis of the lateral sinuses; (*c*) meningitis; (*d*) chronic hydrocephalus.

II. **Distention of the jugular veins** may be due to: (*a*) Intrathoracic pres-

pain, and there may be fever. The etiology is obscure; it may occur with gout or syphilis, or it may be an early expression of Buerger's disease.

(3) **Suppurative Phlebitis:** This results from infections of the walls of the veins by adjoining infected areas. This causes pain and throbbing over the affected vein, edema of the surrounding tissue, fever, chills and other toxic manifestations. Septic emboli may be carried by this infection to distant parts of the body.

Venous Thrombosis

Venous thrombosis may be caused by phlebitis or it may occur as a primary condition; often they occur together. When it is noninfectious and the affected vessel is not large the symptoms are mild When a large vessel is affected the signs are those of venous obstruction If the thrombus is suppurative, signs of local and general infection are prominent. Thrombosis may occur in the lateral, longitudinal and cavernous sinuses. These are usually due to some suppurative lesion in the skull and cause toxic symptoms and local signs. Thrombosis of the central lateral vein is occasionally seen in senile arteriosclerosis and may cause sudden blindness or glaucoma

Glomus Tumors

Glomus tumors are small bluish-red or purplish areas measuring a few mm in size They are found, as a rule, upon the palmar surface of the hand and the plantar surface of the feet, more particularly at the finger tips under the nail beds, on the inner surface of the fingers and on the thenar and hypothenar regions They are formed by convoluted blood vessels made up of peripheral arteriovenous anastomoses surrounded by muscle and epithelial cells They may occur singly or in numbers and are exceedingly painful. The pain is of a burning character and is aggravated by exposure to heat.

Telangiectasia (Angiomatosis)

Telangiectasia is a localized enlargement of the smaller superficial vessels These enlarged vessels may be found in the mucous membranes of the nose, mouth or elsewhere; they may also occur upon the face or other parts of the body. This may be hereditary familial (as described by Goldstein), or secondary caused by local injury or disease, and congenital nonfamilial (Navoid). These lesions have a tendency to cause spontaneous hemorrhages.

Peripheral Circulation Function Tests

In determining the adequacy of the peripheral circulation, various tests may be performed; these often indicate the functional capacity of the capillaries and arterioles. The commoner tests are: (1) The histamine test, (2) surface temperature test; (3) the intradermal saline test; (4) capillary resistance test; (5) plantar ischemic test, and (6) oscillometric readings

(1) **The Histamine Test:** 0.1 cc. of 1:1000 histamine is injected intradermally or by the scratch method (care must be taken not to draw blood by the scratch) in several sites upon the part to be tested Normally a wheal begins to appear over the site of injection at the end of two and one-half minutes and is completed at the end of ten minutes The wheal is generally surrounded by an erythematous area flare.

A delayed reaction usually indicates impaired circulation In severe cases of endoarteritis and in Buerger's disease, a wheal may not form at the site of injec-

tion; the back current is stopped at the jugular valve which transmits the shock above. The jugular pulse also differs from the carotid impulse by its force; thus, in the jugular vein the pulsation is mere undulation, while in the carotid artery it is an active circumscribed impact. The venous pulse of tricuspid regurgitation is positive and occurs synchronously with the apex beat and carotid impulse. It is best seen at the right jugular bulb in the supramastoid fossa where the valve of the vein closes above the bulb. When the valve becomes incompetent, a positive systolic venous pulsation can often be felt upward in the neck.

The regurgitation of blood which is urged upward through the incompetent orifice into the auricle with each right ventricular systole, takes place into the superior vena cava, right innominate and internal jugular veins This jugular pulse may disappear while the patient assumes an upright posture because gravity favors its disappearance. In some cases of tricuspid regurgitation the venous pulse can also be noted on the left side. A venous pulsation may disappear when the myocardium becomes very weak or when the heart rate is extremely rapid. Functional tricuspid insufficiency, particularly when associated with pronounced anemia, may temporarily cause a positive jugular pulsation which occurs synchronously with a soft systolic murmur heard over the mitral area.

Venous Hum

This is a continuous humming or buzzing sound which occurs during the filling of a vein and disappears while the vein empties. Three conditions may produce it.

I. *Anemia, due to the change in the* viscosity of the blood and the increased rapidity of the circulation.

II. *Compression of the jugular veins* due to posture (turning the patient's head), pressure of an enlarged gland, or any other condition that may constrict its lumen.

III. *Tricuspid insufficiency*

Phlebitis

Inflammation of the veins is usually accompanied by pain, inflammatory swellings corresponding to the affected vessel and edema of the affected extremity. It is usually the result of an infection or traumatism

Phlebitis may be divided into three groups: (1) Plastic or noninflammatory phlebitis; (2) thrombophlebitis migrans; (3) suppurative phlebitis

(1) **Plastic Phlebitis:** This may occur after an injury, after surgical operation, in fevers such as typhoid, pneumonia, influenza, during puerperium (phlegmasia alba dolens), in local infections, in gout, in thromboangiitis obliterans, in stasis, and in syphilis. Irrespective of its etiology, the symptoms depend upon the size and extent of the vessel affected, and the degree of collateral circulation established. When the return circulation is grossly affected, swelling, coldness and pain in the extremity may develop and may lead to gangrene.

(2) **Thrombophlebitis Migrans:** This is a condition characterized by the occurrence of local areas of thrombophlebitis in various veins at various intervals. It may affect the superficial veins of the arms and legs or the larger visceral veins. When it affects the pulmonary vessels it may cause signs of infarction and hemorrhage. In the superficial veins it causes localized redness and

lations are absent in occlusive vascular diseases.

X-ray of the Arteries: This may reveal the presence of calcareous infiltration Arteriography has at present a limited field of usefulness. When harmless opaque solutions for intravascular use are found, arteriography and intravenous and intracardiac studies by x-rays should be of greater use.

The Lymphatic System

The lymphatic system consists of the thoracic duct, the right lymphatic duct, smaller lymphatic vessels (lymphatics), tissue spaces, lymph nodes or glands, and a large number of lymphoid cells in various sized groups distributed among all the organs and most of the tissues of the body The function of the lymphatic system is not entirely known The various lymphatic nodes appear to act as filters of the blood plasma, both abstracting from and adding substances to the tissue fluids. The lymphoid glands, among their other functions, are the source of the lymphocytes. The lymph is collected from the various spaces, tissues and organs by the lymphatics which run parallel to the veins. The lymphatics, like the veins en route to the heart, continue to join larger vessels until the largest lymphatic vessels are formed These, the thoracic duct and the right lymphatic duct empty into the left and right large venous trunks which pour their contents into the right auricle and thence into the blood stream.

Diseases of the Thoracic Duct

Obstruction of the thoracic duct by inflammation, tumors or tuberculosis may cause chylous effusion in the pericardium, pleura or peritoneum. The diagnosis of disease of the thoracic duct is not easily made.

Disease of the Lymphatic Vessels

Lymphangitis: Acute lymphangitis occurs as the result of acute local infections. It is characterized by the occurrence of red streaks leading from the infected area towards the regional lymph nodes. The reddened streaks are tender to touch and the lymph nodes are swollen and tender to touch.



Fig. 14—Elephantiasis. (Courtesy of Dr D. Budin.)

Lymphangiectasis: Dilatation of lymphatic vessels usually results from

tion. In vasospastic disease, the histamine reaction may develop slowly. The return of a histamine reaction where it was previously absent denotes recovery.

(2) **Surface Temperature Test:** The surface temperature of a part may be tested in various ways. Ordinary palpation may reveal gross changes in the temperature of various parts; the less obvious changes of temperature may be detected by the aid of various instruments of the dermocouple type such as the dermatherm, the potentiometer, etc. By the aid of these instruments the surface temperature of various parts of the body may be determined when at rest, following exertion, following the application of heat or cold to two similar parts (as parts of both upper or lower extremities), and then the temperature of each is measured and the rapidity with which the temperature of each of the tested parts returns to normal is noted.

The determination of temperature of a part after block anesthesia is an adequate differential point. In total occlusion of the vessels of a part, the temperature does not rise after nerve block, spinal or general anesthesia, but will rise to a considerable degree in the presence of vasospasm. Also, according to Gibbon and Landis,[1] in the normal individual when the upper extremities are immersed in warm water for one hour, the temperature will rise in the lower extremities (or when the lower extremities are immersed the temperature will rise in the upper extremities). In the presence of total occlusion such change does not occur, but in vasospasm a normal reaction usually occurs.

(3) **The Intradermal Saline Test:** This consists of injecting 0.2 cc. of normal saline solution intradermally at various levels of the part to be tested, and noting the length of time required for the absorption of the wheal. In the normal, the wheal may not be totally absorbed within one hour. The disappearing time is considerably reduced in vascular disease and the time increases when the vessels improve.

(4) **Capillary Resistance Test:** This consists of creating a localized erythema and noting the number of capillary hemorrhages in that part. To induce the hyperemia a tourniquet is applied tightly around the arm, a vacuum cup may be applied, or the skin over the bony prominence may be flipped An increased number of capillary hemorrhages denotes diminished capillary resistance. This is found in purpura, scurvy, vitamin C deficiency, various fevers, toxemia, nutritional defects and some of the blood dyscrasias.

(5) **Plantar Ischemia Test (Buerger):** This is performed by having the reclining patient keep his feet elevated at an angle of 90 degrees and extend and flex his feet and toes at the rate of 40 to 60 times a minute for one minute. In the presence of occlusive vascular disease, marked pallor appears upon the sole and toes of the affected foot. Normally no color change is noted.

(6) **Oscillometric Reading:** The oscillometer, or an ordinary sphygmomanometer may be employed to test for arterial pulsation in an extremity. The cuff is applied around the calf of the leg and inflated to a point corresponding a little above the individual's diastolic pulse pressure. The vigor and extent of the oscillations of the mercury column in a mercury instrument or the needle in an aneroid instrument will indicate the patency of the arteries in the leg. Oscil-

[1] Gibbon, J. H. and Landis, E. M: Jour. Clin Invest. 11:5 (Sept) 1932

kin's disease, etc., a definite diagnosis can be made only after a biopsy

Status Lymphaticus: This is a condition in which there is hyperplasia of all the lymph glands of the body including the thymus gland (For details see p 785)

Hodgkin's Disease: This at times is alluded to as lymphadenoma (For details see p 569)

Bone Marrow

The bone marrow begins to develop hemopoietic function at about the middle of fetal life This function continues after birth, supplying the individual with the erythrocytes and the granular leukocytes throughout life.

There are two types of bone marrow: red and yellow. At birth and until near puberty, all the bones are filled with red (cellular, hyperplastic) marrow Thereafter, the red marrow in the long bones begins to change to yellow (aplastic) This is due to the infiltration of fat which replaces the active red cells All the marrow of the long bones except that in the upper end of the femur and humerus becomes yellow by about the age of twenty. In the normal adult, the red marrow is found mainly in the sternum, the ribs, the bones of the skull, the pelvis, the vertebrae and the ends of the humerus and femur The volume of red bone marrow in the adult has been estimated to comprise from 3 4 to 5 9 per cent of the body weight

The yellow marrow of the long bones in the adult may turn pink under conditions in which abnormal increased marrow activity occurs

Microscopic Appearance of Active Bone Marrow: Microscopically, the active bone marrow is seen to consist of a reticular cell mass in the meshes of which lie proliferating granulocytes, red blood cells, and megakaryocytes Traversing this mass is an extensive capillary system, most of which is collapsed

Types of Cells: The histiocyte or hemohistioblast is probably the forerunner of all the blood cells. erythroblastic, myeloblastic, lymphoblastic and monocytic. The primitive red cell, erythrogone, is seen with red cell hyperplasia

The approximate proportions of red cells in the normal marrow are: erythrogones, 0; megaloblasts, 0 to 2 per cent; young erythroblasts, 5 per cent; erythroblasts, 15 to 25 per cent, and normoblasts, 70 per cent

The approximate proportions of white cells in normal marrow are: myeloblasts 1 to 5 per cent; promyelocytes, 2 to 10 per cent; myelocytes, 40 to 60 per cent; metamyelocytes, 20 to 30 per cent, and mature polymorphonuclears, 5 to 10 per cent

Bone marrow may be obtained for study either by puncturing the sternum and aspirating some marrow or by trephining a small portion of bone so as to obtain an intact specimen

Technic for Sternal Puncture: Sternal puncture is feasible only in adults, since the marrow cavity of this bone is poorly developed in children The patient assumes the supine position in bed or on an operating table The skin over the sternum and adjacent area is made surgically aseptic. The favored site for puntcure is the approximate center of the gladiolus, though the manubrium may be used if necessary. After the skin, subcutaneous tissue and periosteum at the site of puncture have been anesthetized with a 1 per cent procain solution, the skin and subcutaneous tis-

obstruction of the larger lymph vessels by scar tissue, carcinoma or other tumors or by infiltration of the vessel walls by inflammation, tuberculosis or syphilis Obstruction of the deeper vessels causes dilatation of a group of smaller lymphatics.



Fig 15—Bilateral elephantiasis (Courtesy of Dr. E. Robertson.)

Elephantiasis: This is a chronic diffuse swelling of one or both legs The extremities are swollen, cool to the touch and do not pit on pressure, or only slightly so. It is due to obstruction of the lymph channels draining the affected part. Elephantiasis may be acquired or congenital. The acquired form results from injury, inflammation, malignancy to the lymphatics or from invasion of the lymphatics by *Filaria sanguinis hominis* (SEE: pp. 752, 1076, 1080)

Milroy's disease or ***Meige's disease:*** This is a familial, hereditary type of elephantiasis.

Unilateral elephantiasis: This usually affects one lower extremity and often also the genitalia; occasionally it develops idiopathically at or about puberty It is more common in females than in males.

Disease of the Lymph Nodes

Lymphadenitis: This may be acute or chronic, generalized or localized.

Acute lymphadenitis: This occurs as a result of local infection associated with lymphangitis It is also associated with some of the acute infections such as rubella, measles, scarlet fever, diphtheria, mononucleosis (glandular fever), etc.

Chronic lymphadenitis: This may occur in pyogenic infections, tuberculosis, syphilis, lymphadenomata, carcinoma, sarcoma, Hodgkin's disease, lymphatic leukemia, status thymicolymphaticus and in Mikulicz's disease

Mikulicz's disease is a slowly developing bilateral painless enlargement of the lacrimal and the salivary glands, *i.e*, parotid, submaxillary and sublingual glands The enlargement is due to hyperplasia of the lymphoid tissue, not to the secretory elements of the salivary gland It is of unknown etiology and occurs only during adulthood

Lymphosarcoma: These may affect any of the lymphatic glands and metastasize by way of the lymphatics to distant organs. The most common primary lesion is in the cervical glands; other sites for primary lesions are the mediastinum, the tonsils, the nasopharynx, the retroperitoneal lymph glands and the lymphoid tissue of the intestine.

Diagnosis: Since cervical adenitis may also be caused by tuberculosis, syphilis, lymphocytic leukemia, Hodg-

SECTION 8

Diseases of the Blood-Forming Organs Associated with Microscopic Changes in the Blood

sue are penetrated with a special needle which is inserted vertically with the bevel toward the patient's chin. As the periosteum is reached, it is penetrated with a rotatory boring motion until the bone is grazed The needle is then tilted downward until it forms an acute angle of about 30 degrees with the surface of the body; then, with another quick, short rotatory boring thrust, the needle is forced through the anterior plate of the sternum. As the needle enters the marrow cavity of the bone, the sense of resistance suddenly ceases. The ball guard is then adjusted on the outer needle to prevent it from slipping, and the stylet is removed from the inner needle to which a syringe containing about 1 cc. of normal saline is attached and a small portion (1 cc. or less) of the marrow is slowly aspirated. The amount of force required to aspirate the marrow indicates its density.

Indications for Sternal Puncture: Sternal puncture is a safe and comparatively easy procedure after one has mastered the technic on the cadaver. Examination of the marrow is a valuable aid in the diagnosis of acute leukemia, aleukemic leukemia, multiple myeloma, and some of the obscure anemias. It is also helpful in the diagnosis of malaria, particularly the falciparum type, and of leishmaniasis and Gaucher's disease. In most instances, simple sternal puncture is usually satisfactory. Occasionally, however, it becomes necessary to resort to trephine biopsy of the sternum, a rib, or a long bone.

Blood Transfusion Through Sternal Puncture: Sternal puncture is also a valuable procedure for blood transfusion, and infusion of plasma and normal saline solutions in patients whose veins cannot be used

Blood Transfusion by Way of Bone Other Than Sternum: In infants and young children where sternal puncture is not feasible, the upper portion of the tibia or lower portion of the femur at the metaphysis may be used for transfusing blood or other fluids When the tibia is used, the needle is to be inserted at a point 2 cm. below the condyle. If the femur is used, the needle should be inserted above the external condyle. The needle should always be pointed toward the diaphysis of the bone so as to prevent injury to the epiphyseal cartilage.





b





c



Stages in Formation of Red Blood Cells

a Megaloblasts b Older megaloblasts with condensation of nuclei c, Normal erythrocytes (Hull, Wright & Eyl's "Medical Nursing" F A Davis Co, Philadelphia, Pa.)

(Wright's stain). The granules may appear either uniformly or irregularly distributed throughout the cell; they may appear in several groups in the cell or form a ring around the cell circumference. This is found in severe primary and secondary anemia, especially that of lead poisoning; also in malaria and leukemia, but not in aplastic anemia.

Embryonic Cells—*Microcytes* are erythrocytes smaller than normal; they are found in hypochromic, hemolytic and other forms of anemia associated with oligochromemia.

Macrocytes are erythrocytes larger than normal; they are found in certain forms of anemia, *viz.*, the hyperchromic anemias.

Normoblasts are nucleated red cells of normal size and normal staining power. They each have a small deeply staining nucleus which may be round, lobed or clover leaf shaped. Occasionally they may be broken up into two or three nuclei. These are seen in severe forms of anemia.

Megaloblasts are nucleated red cells larger than normal, each containing a large nucleus and polychromatophilic cytoplasm; they are found in some types of severe anemia, especially in pernicious anemia.

Microblasts are nucleated red cells smaller than normal; they are found in some forms of severe anemia.

Poikilocytes are deformed or irregularly shaped red cells; they may be oval, pear-shaped, elliptical, club-shaped or any other form; they are found in the blood of severe types of anemia Poikilocytosis occurs in conjunction with anisocytosis (variation in size).

Reticulocytes are very young or immature red corpuscles containing a coarse network of granular fibrils or filaments. Their presence in the blood stream *is an indication of blood regeneration*. Normally in adults they are found to be less than 1 per cent, and *in young infants from 2 to 4* per cent In some of the blood diseases; *i e*, pernicious anemia, hemolytic jaundice, etc., when blood regeneration is active, a high percentage of reticulated red blood corpuscles appear in the blood stream Reticulocytes, when present in the blood, are discovered only by the "vital staining" method and are not found by the ordinary dry slide staining method

Embryonic red cells are found in all types of severe anemia where the destruction of blood cells is faster than their manufacture. The blood-making organs, in order to meet the demand for more cells than they can supply, throw into the circulation a number of unfinished (embryonic) erythrocytes

The Blood Dyscrasias

The diagnosis of the various blood diseases associated with changes in the number and type of the red and white cells, the hemoglobin percentage and the number of platelets is usually made by laboratory studies of the freshly drawn blood. Many of these diseases, in addition to characteristic hemograms, also show definite physical signs and clinical symptoms.

Symptoms such as weakness, tingling of the extremities, headache, digestive disturbances, glossitis, certain nervous manifestations and cardiac palpitation are usually found in most of the blood dyscrasias

Physical signs such as pallor, either of a lemon yellow tint, an ashen gray or a bloodless hue, are found in various forms of anemia. Subcutaneous and submucous membrane hemorrhages are

found in severe anemias, leukemia and purpura Enlarged lymph glands, a large spleen and liver are found in the leukemias, in a few of the anemias and in Hodgkin's disease. A functional heart murmur may result from impoverished blood, and occasionally the presence of an organic murmur may give a clue to the cause of anemia.

The Anemias

Some of the anemias are primary or idiopathic, others are secondary

A *primary anemia* is one in which no etiologic factors are discoverable. Pernicious anemia is considered a primary hyperchromic macrocytic anemia, and chlorosis is considered as a primary hypochromic microcytic anemia.

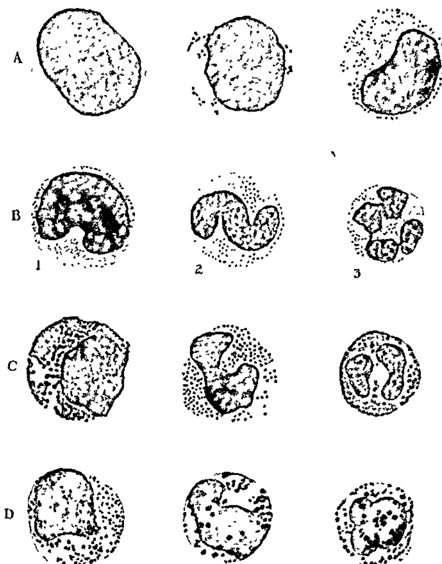
Secondary anemias are so called when a definite etiology is discoverable and the anemia is a development as a consequence of, or in the course of a definite pathologic entity, such as carcinoma, bacterial or parasitic invasion and defective nutrition. Secondary anemia is usually accompanied by a considerable weight loss In the primary anemias, the loss of weight is not marked.

The anemias may be classified as: Macrocytic hyperchromic anemia; microcytic hypochromic anemia; hemorrhagic anemia; aplastic anemia; hemolytic anemia, etc.

Macrocytic Hyperchromic Anemia

Macrocytic hyperchromic anemia is characterized by a *low total red cell count* in which are found many megalocytes and macrocytes containing a high hemoglobin content In severe cases there may be various types of red cells that indicate aplasia or hemolysis, often both The color index is usually above *one.* The gastrointestinal findings generally associated with this type of anemia are achylia gastrica or a very low hydrochloric acid content; various signs of indigestion, such as epigastric distress, belching, flatulency, diarrhea or constipation in most cases; and, in a fairly large number of cases, glossitis or burning of the tongue. In some cases there may be associated definite pathologic lesions in the stomach, bowel, liver or pancreas, while in others there may be a total absence of any organic lesions in the digestive tract.

The explanation of the occurrence of macrocytic hyperchromic anemia is based on the theory of incomplete maturation of the erythrocytes In health the formation of an adequate number of red corpuscles is attributed to the presence of a hematinic maturing principle in the blood stream This principle, according to Castle and his associates, is stored in the liver. It is formed by a combination of the "intrinsic factor" found in normal gastric juice which is secreted by the gastric mucosa or by the pyloric and Brunner's glands, and an "extrinsic principle" which is taken into the gastrointestinal tract with food A deficiency of the hematinic maturing principle in the blood stream for the use of the bone marrow, will prevent the red corpuscles from maturing beyond the megaloblastic stage, thereby causing this type of anemia. The red bone marrow is increased in quantity and is loaded with megaloblasts which contain large amounts of hemoglobin A comparatively small number of these megaloblasts progress beyond this stage, and develop into macrocytes (large hyperchromic erythrocytes). The deficiency of the hematinic principle may be brought about in six or more ways:



STAGES OF MATURATION OF LEUKOCYTES

A, Lymphocytes B, Polymorphonuclear neutrophilic leukocytes: Metamyelocyte 1, Juvenile 2, Stab cell 3, Segmented cell C, Eosinophilic leukocytes D, Basophilic leukocytes (Hull, Wright & Eyl's "Medical Nursing" F A. Davis Co Philadelphia, Pa.)



VARIOUS FORMS OF ERYTHROCYTES
(Anders and Boston, W. B. Saunders Co.)

1. Defective secretion of intrinsic factor due to disease or atrophy of the glands that secrete this principle.

2. Absence or defective intake of the extrinsic factor.

3 Defective absorption from the intestinal tract of the intrinsic or the extrinsic factors, though both may exist in sufficient quantities

4. Defective storage of the hematinic principle in the liver and other organs

5 Failure of the formation of a hematinic principle because of faulty interaction between the intrinsic and extrinsic principles

6. Failure of the bone marrow or other factors concerned with the production and maturation of the red corpuscles to utilize the hematinic principle

Primary Pernicious Anemia (Addison-Biermer Anemia) · This is a hyperchromic macrocytic type of anemia of unknown etiology, and is characterized by a definite symptomatology and characteristic blood findings

Addison, in 1855, described this disease as follows:

"It makes its approach in so slow and insidious a manner that the patient can hardly fix a date to the earliest feeling of that languor which is shortly to become so extreme The countenance gets pale, the whites of the eyes become pearly, the general frame flabby rather than

exertion, with an uncomfortable feeling of faintness or breathlessness in attempting it; the heart is readily made to palpitate; the whole surface of the body presents a blanched, smooth and waxy appearance; the lips, gums and tongue seem bloodless, the flabbiness of the solids increases, the appetite fails, extreme languor and faintness supervene, breathlessness and palpitations are produced by the most trifling exertion or emotion; some slight edema is probably perceived about the ankles; the debility becomes extreme—the patient can no longer rise from bed; the mind occasionally wanders; he falls into a prostrate and half-torpid state, and at length expires; nevertheless, to the very last, and after a sickness of several months' duration, the bulkiness of the general frame and the amount of obesity often present a most striking contrast to the failure and exhaustion observable in every other respect."

Among the symptoms in this disease, aside from progressive anemia and retention of subcutaneous fat, are achylia gastrica, glossitis, or pain in the tongue, general weakness, dyspnea, headache and spinal cord symptoms

Physical Signs: Inspection The patient is usually well nourished, has a waxy lemon yellow appearance; the mucous membranes are pale, the conjunctivae pale, bluish and icteroid; the face puffy; the ankles somewhat swollen; the tongue pale and smooth, resembling the tongue of a fowl.

On *palpation*, the skin has a soft nonelastic feel, the apex beat is barely palpable; the spleen, usually enlarged *Percussion* shows no definite signs *Auscultation* may reveal a hemic murmur over the body of the heart.

Other Signs. In the early stages, the patellar reflex may be exaggerated and as the disease progresses this reflex disappears The gastric secretion presents an absence of free hydrochloric acid; the urine is of low specific gravity, dark in color and contains urobilin.

Blood examination will reveal a great reduction in the number of red corpuscles, usually less than two million, a large number of which are macrocytes; in severe cases there are poikilocytes, normoblasts and megaloblasts. The color index is comparatively high, always above one. The blood platelet count is seldom over 100,000 Leukopenia is the rule The polymorphonuclear cells are

reduced. The lymphocytes are increased in number as are also the myelocytes The plasma is reduced in quantity. An indirect Van den Bergh reaction is above 0 75 The reticulocytes are usually absent. When treated with adequate doses of liver or ventriculin or during a remission the reticulocytes appear in large numbers in the blood stream

While remissions in the severity of the blood picture will occur when treated with liver, the achylia gastrica and the neurologic changes are not markedly improved by treatment I like to think of pernicious anemia as a disease of unknown etiology which equally affects the three important systems of the body, namely, the blood-making organs, the digestive system and the nervous system In some patients the digestive system is the first to be affected. Achlorhydria may develop months or years before the other systems show evidence of disease In others, the first system to be affected is the nervous system, and neurologic manifestations may precede the defects shown by the other systems by months or years, while in still others, the anemia is the first sign to be noticed. Occasionally all three systems are simultaneously affected

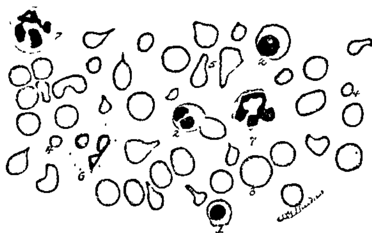
Tropical Megalocytic Anemia: This is probably a deficiency anemia. It occurs in the tropics, often among the natives of India It is characterized by weakness, pallor, digestive disturbance, edema of the ankles, puffiness of the face, low blood pressure, hemic murmurs and occasionally by glossitis. The blood picture reveals a great reduction in red cells and a comparative increase in the hemoglobin percentage. Macrocytosis and anisocytosis are marked. There may be a slight leukocytosis or a normal count The platelets are reduced; the indirect Van den Bergh reaction is normal (below 0 75); hypochlorhydria or a normal acidity may be present, seldom an achylia. This condition should be differentiated from pernicious anemia which it closely resembles. The absence of poikilocytosis, polychromasia and the normal indirect Van den Bergh (below 0.75), the presence of gastric acidity and the absence of urobilinogen are in favor of tropical megalocytic anemia.

Secondary Hyperchromic Macrocytic Anemia: This may occur in tropical sprue, idiopathic steatorrhea, and infestations with diphyllobothrium, in vitamin B deficiency and exposures to large doses of x-rays or radium. It may also be found at times in malignancy of the stomach or colon, in regional ileitis, or it may follow gastrectomy or other operations upon the gastrointestinal tract. Occasionally it may be found in myxedema, malarial cachexia, after prolonged hemorrhage, during pregnancy and in early childhood. In these cases, in addition to the secondary anemia of the hyperchromic macrocytic type showing a high color index, there are found either an achlorhydria or a hypochlorhydria, and various gastrointestinal disorders, and nervous manifestations in association with the signs and symptoms of the primary lesions.

The macrocytic hyperchromic anemia often responds to liver therapy, particularly so when the etiologic factor is removable.

Microcytic Hypochromic Anemia

Microcytic hypochromic anemia is characterized by a *reduction of the hemoglobin content* within the red corpuscles. The red corpuscles are usually reduced in number and often in size. The number of red corpuscles in this type of anemia



Blood of Pernicious Anemia
(Anders and Boston, W B Saunders Co)



Blood of Chlorosis
(Anders and Boston, W. B. Saunders Co.)

seldom, if ever, falls to the very low level reached by cases of macrocytic anemia. The red cell development is arrested at the level of the erythroblastic stage and the cells are released into the blood stream as erythrocytes only when sufficient iron and possibly other substances are available for the formation of an adequate amount of hemoglobin to fill them.

This type of anemia may be considered as an iron deficiency anemia and may be produced: (1) By a lack of iron in the food; (2) by the inability of the digestive tract to separate the iron from iron-containing food; (3) by the inability of the digestive tract to transmit its ingested iron to the blood stream, and (4) by the inability of the blood-making organs to utilize iron. The diseases associated only with hypochromic anemia are:

Chlorosis (Green Sickness): This is a primary microcytic hypochromic type of anemia of unknown etiology found in young women. It is characterized by oligochromemia.

Symptoms and Diagnosis: The patient is usually fat. The skin has a pale greenish tinge. The mucous membrane is pale. In some instances, the cheeks may have a reddish flush, particularly so on exertion or during emotion (*Chlorosis rubra*). Dyspnea and palpitation are well marked and there is a tendency toward syncope and general weakness; the face and ankles are puffed and a hemic murmur may be heard at the apex or base.

Blood Examination: The red corpuscles are not greatly reduced in number; the greatest reduction, however, is found in the percentage of hemoglobin A red cell count of four million with only 40 per cent of hemoglobin is not uncommon. In severe cases nucleated as well as irregular shaped red corpuscles may be found in the blood. The leukocytes may be slightly increased in number. The lymphocytes are normal and the blood plates are usually increased. The reticulocytes are within normal limits. Gastric disturbance, such as indigestion, constipation and hypochlorhydria are accompanying signs

Idiopathic Hypochromic Microcytic Anemia (simple achlorhydric anemia): This is a chronic type of anemia found chiefly in women of the menopausal age.

Symptoms and Physical Signs: There is easy fatigability, weakness, nervousness, dyspnea, cardiac palpitation and digestive disturbances. Soreness of the tongue is a frequent complaint and is often associated with a geographic tongue. The patients are usually thin and have a muddy yellowish or grayish complexion. The sclerae are bluish white; the hair is thin, lusterless, and there is early graying. The fingernails are brittle, break easily and are often concave. The spleen is nearly always enlarged. Edema of the feet and often of other parts of the body occurs in severe cases.

Blood Examination: The erythrocytes may range in number from 4,000,000 to 2,000,000 or lower; there is often marked microcytosis with *hypochromia* The color index may range from 0 7 to 0 4. Anisocytosis and poikilocytosis occur in severe cases. Free hydrochloric acid is absent in most cases. Treatment with adequate doses of iron and feeding meat and green vegetables usually causes marked improvement of the anemia

Plummer-Vinson Syndrome: This condition is associated in the majority of cases with hypochromic microcytic

anemia; it is characterized by the occurrence of dysphagia and glossitis. It occurs most frequently in women The symptoms are weakness, fatigue, gastric distress, difficulty in swallowing solid food, painful tongue, and a sensation of having a lump in the throat or retrosternally. There are also various nervous manifestations such as irritability, headache, and sleeplessness.

The physical **signs** present are pallor of the skin and mucous membrane, dryness of mouth and pharynx; dry, brittle and concave fingernails, and tachycardia

Blood Examination: The blood presents a low cell count, and very low hemoglobin content, the color index may be below 0.5 Hypochromasia is nearly constant. The number of leukocytes vary; they may be very low or somewhat increased The free hydrochloric acid content of the stomach may be low or absent.

Other Conditions Presenting Hypochromic Microcytic Anemia: Hypochromic microcytic anemia may also occur in premature infants, and in mature infants if on an exclusive cows' milk diet. It may also occur during pregnancy and lactation This type of anemia may at times be found in idiopathic steatorrhea (Gee's disease, celiac disease, nontropical sprue), in gastric carcinoma and other lesions of the gastrointestinal tract, and it may follow operations upon the stomach or intestines Occasionally these diseases may show, instead of a hypochromic anemia, a hyperchromic type of anemia. Hypochromic anemia is also often found in association with hookworm disease, pellagra, chronic wasting disease, chronic or prolonged hemorrhages from any part of the body, prolonged suppuration, chronic cardiovascular diseases, nephritis, nephrosis and various systemic diseases.

The symptoms, in addition to weakness and pallor, depend upon the underlying disease.

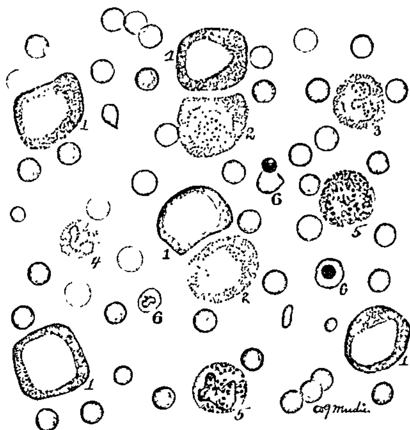
Hemorrhagic Anemias

Anemias due solely to hemorrhage, irrespective of their cause, present definite symptoms which, however, are modified by the amount of blood lost

Anemia resulting from bleeding secondary to an underlying abnormality will present symptoms of both the anemia and the primary disease.

Acute Hemorrhage: In this condition the anemia is of the hypochromic or normochromic type A sudden very large hemorrhage of three or four pints (1500 to 2000 cc) may cause death in a few hours. During the bleeding or soon thereafter, if death does not occur, there are usually signs of shock, such as faintness, syncope, sighing, partial blindness, restlessness, anoxemia, rapid shallow breathing, and thirst. The patient is pale, has a cold clammy skin and may be sweating profusely. The temperature is subnormal The pulse is rapid and thready and the blood pressure is low The blood picture may at first be deceptive Because of the shock, as pointed out by V. H Moon, there occurs hemoconcentration, which shows an abnormally high erythrocyte count and with a high hemoglobin content Later the blood will show a low red cell count, low hemoglobin, immature red cells, leukocytosis and an increased platelet count

Chronic Hemorrhage: When due to frequent epistaxis, hemorrhoids, menorrhagia and from pulmonary, bronchial or gastrointestinal pathology, the severity of the anemia will depend upon the chronicity of the bleeding and the amount



Blood of Myelogenic Leukemia
(Anders and Boston, W B Saunders Co)

nicious anemia and are also encountered in persons suffering from prolonged hemorrhages such as epistaxis, bleeding hemorrhoids, and bleeding gastric ulcers that continue to ooze blood over an extended period of time.

Hemolytic anemia may also result from the ingestion of certain gases, organic or inorganic poisons, various drugs and from bacterial invasions Hemolytic anemia often occurs idiopathically. The excessive red cell destruction or hemolysis is characterized by the presence of hemolysins in the blood, by a positive indirect Van den Bergh reaction, and by the presence of urobilin and urobilinogen in the urine and feces Jaundice may occur in varying degrees of severity The conjunctivae are stained less deeply than is the skin, and the feces are dark brown, which contrasts with the clay-colored stools of obstructive jaundice.

Hemolytic anemia may be congenital or acquired Hemolytic anemia differs from aplastic anemia in that in hemolytic anemia there is an abnormally rapid destruction of blood cells so that embryonic cells enter the blood stream in large numbers and in all stages of development and the bone marrow hypertrophies because of excessive function In aplastic anemia there is primarily an inability of the bone marrow to form cells, therefore there are no embryonic cells in the blood stream to replace destroyed cells All the red cells in the blood are of the mature type.

Acute Hemolytic Anemia (acute hemolytic anemia of Lederer): The onset is sudden with high fever, headache, sore throat, hematuria, diarrhea, vomiting and abdominal pain and occasional epistaxis. It may occur in adolescents and young adults The individual is pale and may show various degrees of jaundice (SEE: p. 603). In severe cases there may be hemorrhages from the mucous membranes and in the skin. The liver and spleen are moderately enlarged.

The blood shows a red cell count between 1,000,000 to 1,500,000 in which are found numerous microcytes, macrocytes, myeloblasts, myelocytes and nucleated red cells. The reticulocyte count is high and may reach 50 per cent or higher. The hemoglobin percentage may vary from 0 5 to 1.5 per cent or it may be 1. The Van den Bergh reaction is positive indirect (above 0 75 units).

Subacute and Chronic Hemolytic Anemia: This may occur in conjunction with severe debilitating diseases in children or adults The blood may show macrocytes, megaloblasts and a high color index. Urobilin in the urine is increased and the Van den Bergh indirect is above 0 75.

Various Other Types of Hemolytic Anemia; *Acholuric Jaundice* (hemolytic icterus, familial hemolytic jaundice with splenomegaly, hemolytic icteroanemia) · This is a chronic congenital or acquired familial blood dyscrasia, manifesting increased blood destruction

Physical examination reveals a generalized jaundice of the skin and mucous membranes. The spleen is usually greatly enlarged The characteristic *blood findings* are as follows Red corpuscles from 1,500,000 to 3,500,000 exhibiting increased fragility and variation in the size of the cells (anisocytosis), polychromasia, nucleated red cells with a preponderance of microcytes; pronounced recticulocytosis may be discovered by the "vital staining" method; the hemoglobin varies from 0.6 to 0 9 per cent; the leukocyte count may be normal or slightly increased. The *feces* are very dark and the *urine* is bile stained.

of blood lost. The anemia is generally of the hypochromic type so that the hemoglobin percentage is low, the number of red cells may vary from 4,000,000 in mild cases to 2,000,000 or less in severe cases, and a large number of these are *microcytes*.

Aplastic Anemia

Aplastic anemia is a severe progressive anemia of unknown origin, characterized by a degeneration of the bone marrow (which often appears yellow and fatty) and a failure of blood formation. It may be primary or secondary; acute or chronic.

Acute Aplastic Anemia: This is a primary, rapidly fatal disease and is characterized by rapid progressive anemia, marked tendency to hemorrhages into the skin and mucous membranes and paroxysms of fever.

Symptoms and Diagnosis: The skin is yellowish; the spleen is not enlarged; there is a marked tendency to hemorrhages into the skin and mucous membranes. The blood shows extreme oligocythemia but shows no embryonic cells. The erythrocyte count may be as low as 1,000,000 or lower. Nucleated red cells are practically absent as are also macrocytes, poikilocytes and reticulated erythrocytes. The platelets are reduced in number and leukopenia is marked, often as low as 2000. The polymorphonuclear cells are greatly decreased in number, while the lymphocytes are relatively or actually increased (from 80 to 90 per cent). This disease is caused by a failure of the blood-making organs to manufacture red corpuscles

Chronic Aplastic Anemia: This may be primary or may develop during the course of severe infections, systemic diseases and in pernicious anemia, when the constant demand upon the blood-making organs has so exhausted them as to produce an aplasia. The clinical picture of this form is slowly progressive but eventually resembles the acute type.

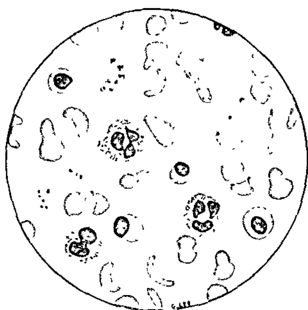
Secondary Aplastic Anemia Either Acute or Chronic: This may occasionally be associated with chronic sepsis, severe forms of nephritis, and may also be caused by poisoning with arsphenamine, benzol, arseniobenzol, dinitrophenol, mercury, silver and gold and by overexposure to x-rays and particularly to radium.

The ***symptomatology*** of the secondary form is similar to the primary form

The ***blood*** shows severe anemia, absence of embryonic cells, marked leukopenia, and thrombocytopenia. The bleeding time is prolonged. Coagulation time is normal Clot retraction is poor. Hemorrhages may occur from the mucous and serous membranes, subcutaneously and from the internal organs. Aplastic anemia is to be differentiated from pernicious anemia, acute leukemia, purpura and agranulocytic angina

Hemolytic Anemia (Hyperplastic Anemia, Hemolytic Icterus)

This type of anemia is characterized by rapid destruction of the red corpuscles and, in order to keep the corpuscular elements in the blood as nearly normal as possible, the bone marrow hypertrophies and sends out embryonic cells (immature red cells) so that normoblasts, microcytes, macrocytes, megalocytes, poikilocytes and a large number of reticulocytes are found in the circulating blood. The hemoglobin percentage is low in most cases, though a high hemoglobin content may occur in some types These blood findings may occur in per-



REPRESENTATIVE SMEAR FROM PATIENT WITH SICKLE CELL ANEMIA

Note sickling of red blood cells and presence of nucleated erythrocytes (Hull, Wright & Eyl's "Medical Nursing" F A Davis Co, Philadelphia, Pa)



Splenectomy is often a satisfactory form of treatment.

Conditions Causing Hemolytic Anemia: Hemolytic anemia may also occur in the following conditions:

Acute and Chronic Malaria: The anemia is usually of the hypochromic type. The red cells show anisocytosis, poikilocytosis, polychromasia and an increase in the reticulocytes. Moderate leukocytosis or leukopenia may be present with an increase in the monocytes. The icterus index may range from 15 to 30.

Oroya Fever (Bartonella bacilliformis infection, Peruvian wart): This is an acute fever indigenous to South American mountainous regions. The organisms invade the red corpuscles and the endothelial cells of the lymph nodes The anemia is severe and is megalocytic in type. The red cell count may be as low as 1,000,000. The leukocytes may vary from 15,000 to 20,000; the majority are immature polymorphonuclears The Van den Bergh is indirect positive. The icterus index may be quite high

Clostridium Aerogenous Capsulatus (Welch's gas bacillus) *Infection* The anemia is ushered in rapidly, often within a few hours; all types of immature cells and cells in various stages of destruction are found in the peripheral blood; leukocytosis may be as high as 50,000. In addition to the anemia there are various degrees of jaundice.

Other severe infections such as typhoid, typhus, syphilis, etc., and also suppurations may occasionally be complicated by this type of anemia.

Chemical poisons such as lead, arsenic and its compounds, arseniurated hydrogen, phenylhydrazine, pyridine, sulfanilamide, sulfapyridine, amidopyrine, cinchophen, potassium chlorate, the nitrates, methylchloride and others of that group may produce various stages and degrees of hemolytic anemia. In these cases, in addition to the anemia and abnormal red cells, there are also various degrees of hemoglobinuria, jaundice and a positive indirect Van den Bergh reaction.

Sickle Cell Anemia: Sickle cell anemia is classified as a hemolytic anemia of unknown origin having a familial tendency. It occurs chiefly in full-blooded negroes, mulattoes, or in those of milder dilutions of negro blood. Several cases were also reported in Caucasians Two stages are recognized:

1. The *latent stage* in which there are few if any constitutional symptoms and where a blood examination alone will reveal the characteristic picture.

2. The *active stage* which is characterized by extreme weakness, dyspnea, abdominal pain with nausea and vomiting, pain in the muscles and joints and ulcers on the legs

Physical Signs: The physical examination will reveal a poorly nourished and poorly developed anemic negro with a greenish yellow discoloration of the sclerae, enlarged lymph glands, large liver, small or impalpable spleen, though at times it may be enlarged, and ulcerations of the legs often accompanied by edema of the ankles. The heart, lungs, kidneys and digestive tract show the usual signs of grave anemia

The Blood Picture reveals a great decrease in the red corpuscles and hemoglobin and the presence of poikilocytosis, polychromasia, anisocytosis, and the characteristic crescent-shaped red corpuscles, the sickle cells The reticulocytes are increased in number during the regenerative periods Leukocytosis from 15,000 or 25,000 is common and myelocytes are present. The blood serum is often of a decided yellow color.

Paroxysmal Hemoglobinuria: This condition is ushered in with a chill and rise in temperature often following exposure to cold. There may be diarrhea, vomiting, and pain in the back and in the extremities. The skin becomes somewhat jaundiced. The urine is dark and contains hemoglobin and methemoglobin. The blood shows marked anemia of the hypochromic type with immature cells and increased reticulocytes. Many of these patients show positive Wassermann and Kahn reaction.

Banti's Disease (Banti's syndrome, hepatolienal fibrosis, hepatosplenic cirrhosis): This is a disease of unknown etiology occurring in young adults in whom there is enlargement of the spleen and the liver, distention of the venous system, hematemesis, melena and, in late stages, ascites.

The Blood Picture is that of a severe hypochromic anemia. The red cell count may vary from 3,500,000 to 2,500,000, and the hemoglobin may be about 50 per cent. Reticulocytes may be absent except soon after a hemorrhage when a moderate reticulocytosis may be found. Leukopenia with a low polymorphonuclear count is the rule; occasionally, particularly after hemorrhage, there may be a leukocytosis The platelets are somewhat reduced in number. The Van den Bergh reaction may show an increased indirect in the early stages and a positive direct in late stages (SEE: pp. 612 and 623).

Cooley's Anemia (*Erythroblastic anemia, Thalassaemia: Mediterranean fever*): This is a disease that becomes manifested during infancy; it is characterized by a yellowish pallor, mongoloid facies, thickening of the cranial and malar bones and great enlargement of the spleen with some enlargement of the lymph nodes and of the liver.

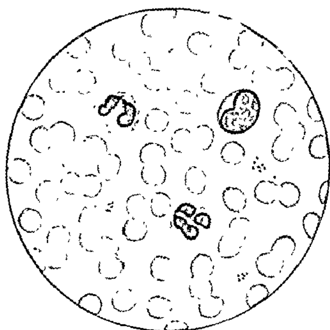
The Blood Picture shows a severe anemia of the leukoerythroblastic type. The red cell count may be below 2,000,000 per cmm. and there are large numbers of nucleated red cells (erythroblasts), many macrocytes and microcytes and anisocytosis. The hemoglobin may vary from 30 to 10 per cent.

Leukoerythroblastic Anemia (myelopathic anemia, osteosclerotic anemia): In this type there are found primary erythroblasts, megaloblasts, normoblasts and hemocytoblasts. There are also present in the blood stream immature white cells of the myeloid type.

This type of anemia is found in carcinomatosis affecting bone, myelosis, marble-bone disease and Cooley's erythroblastic anemia. The presence of immature red cells and the scarcity of hemoglobin indicate aplasia of the blood-forming organs.

Erythroblastosis Fetalis: This is a congenital erythroblastic anemia occurring in the newborn It is believed to be caused by the mother's Rh negative blood mixing with the Rh positive blood cells of the fetus by way of the placenta. It occurs chiefly as icterus gravis neonatorum, congenital anemia of the newborn, and congenital hydrops fetalis. The factors common to these is a severe anemia having a red cell count of 1,000,000 or less with a large number of nucleated red cells and widespread extramedullary erythropoiesis.

Icterus Gravis Neonatorum: This is a congenital severe anemia of the hyperchromic type, showing a large number of immature red cells and a pronounced reticulocytosis; there is marked



NORMAL SMEAR SHOWING TWO POLYMORPHONUCLEAR LEUKOCYTES, ONE LYMPHOCYTE, BLOOD PLATELETS, AND ERYTHROCYTES

(Hull, Wright & Eyl's "Medical Nursing" F A Davis Co, Philadelphia, Pa)

Blood Disease Presenting an Increase in the Number of Red Corpuscles

Polycythemia Vera (Erythremia, Vaquez-Osler Disease)

Polycythemia vera is a chronic blood disease characterized by an increase in the number of red corpuscles, a reddish purplish discoloration of the skin and splenic enlargement.

Symptoms and Diagnosis: Vertigo, headache, buzzing in the ears, fatigue, blurring of vision, paresthesia, mental apprehension and gastrointestinal disturbances are symptoms of this disease

Physical Signs: The skin, particularly of the face, neck, upper chest and hands, presents a reddish cyanosis; the conjunctivae are injected and the retinal vessels are distended, often causing hemorrhage Venous enlargement is observed upon the cheeks, nose and other parts of the body. Hemorrhages in the lungs, brain, kidney and epistaxis are common. The spleen is enlarged and firm to the touch

The Blood Picture: The volume is increased. Erythrocytes may be 7 million to 15 million; hemoglobin, 110 per cent or higher, though the color index is comparatively low. Leukocytes are usually of normal count Bleeding and clotting time are about normal

Erythremia may occur in conditions other than polycythemia vera as a result of blood concentration It is found in congenital heart disease of the right ventricular shunt variety (pulmonary stenosis, patulous foramen ovale), in Ayerza's disease and in dehydration due to diarrhea, excessive sweating and vomiting. It also occurs in chronic emphysema, in people living at high altitude, and in chronic cyanosis. The absence of a large spleen and large retinal vessels, and the presence of such signs as will identify the underlying cause of the cyanosis and polycythemia are differential features to be considered in the diagnosis

Blood Diseases in Which the Plasma and Platelets are Chiefly Affected

Purpura

Purpura is a condition characterized by hemorrhages into the skin and mucous membrane, and is probably caused by some alteration in the clot-forming substances in the blood. It may be primary or secondary.

Primary Purpura: *Simple purpura* is recognized by the occurrence of purpuric spots, chiefly in the lower extremities.

Peliosis Rheumatica (arthritic purpura, Schoenlein's disease): Purpuric spots are distributed over the extremities or trunk and are associated with tenderness, swelling and pain of several joints, accompanied by fever.

Henoch's Purpura (visceral purpura): In this form of purpura skin lesions such as erythema multiforme, purpuric spots, urticaria and angioneurotic edema occur in association with extreme intestinal disturbances, such as colicky pain, vomiting, diarrhea and melena. Enlargement of the spleen is usually present and acute nephritis is a frequent complication.

Idiopathic Thrombocytopenic Purpura (*purpura hemorrhagica* [morbus maculosus of Werlhof]): This form is characterized by bleeding from the mucous membranes of the nose, mouth, stomach, bowels, kidney, bladder and uterus. Cutaneous hemorrhages, either large or small, and hemorrhages in the brain occur frequently. Bruising of the skin or breaking it with a needle or any

jaundice, fragility of the long bones and a tendency to hemorrhage (SEE: p. 604).

Congenital Anemia of the Newborn: This presents a severe anemia and a large liver and spleen; jaundice may or may not be present. The anemia is of the hyperchromic type; reticulocytosis is present in the early stages and disappears later.

Congenital Hydrops Fetalis: This shows an anemia of a severe hypochromic type in which there is present immature red and white cells; the nucleated red cells occur in large numbers. In addition to the anemia, there is a generalized anasarca, with effusions in the serous sacs and a large liver and heart.

Syphilis Hemorrhagica Neonatorum: This occurs in congenital syphilis. Several days after birth extensive subcutaneous bleeding from the mucous membranes and from the navel are apparent. It is accompanied by deep jaundice.

Morbus Maculosus Neonatorum: Fatal bleeding may occur from the various viscera and mucous surfaces. It is accompanied by a rise in temperature and hematogenous jaundice. Septic infections, trauma during birth and eclampsia in the mother are among the conditions that may cause fatal hemorrhages in the newborn.

Werlhof's Disease (*essential thrombopenia, purpura hemorrhagica, thrombocytolitic purpura, hereditary hemorrhagic thrombosthenia, purpura thrombopenica homogenia, hemogenic syndrome*): This is a hemorrhagic diathesis in which there is a great reduction in the number of blood platelets. Werlhof's disease is characterized by the spontaneous occurrence of hemorrhages into the tissues and from the tissues at indefinite intervals and without any apparent cause. A tight tourniquet applied around an extremity will in 10 or 15 minutes produce subcutaneous punctate hemorrhages. The application of dry heat to the skin or the tapping of a bony surface with a percussion hammer may produce ecchymotic areas.

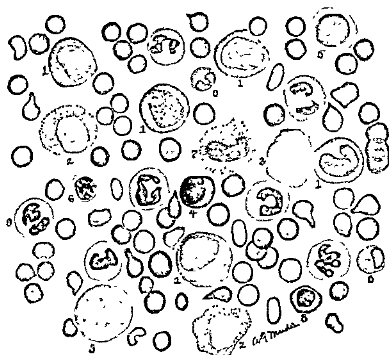
Blood Picture: The characteristic *blood* findings are a great reduction in the blood platelets associated with a variation in their size. The bleeding time is increased. Splenic enlargement is often present.

Von Jaksch's Anemia (anemia pseudoleukemia infantum) This designates a type of blood impoverishment classifiable as secondary anemia. It occurs chiefly in young infants of rachitic tendencies.

Physical Examination: This reveals a pale, somewhat flabby, restless child, having a large abdomen and palpable lymph glands. The liver is enlarged, smooth and not very firm to touch Its edge is well rounded. The spleen often becomes enormously enlarged. As a rule the spleen has twice the enlargement of the liver.

The blood picture is that of secondary anemia plus leukocytosis. The red corpuscles number from 2,000,000 to 3,000,000 to the cmm.; hemoglobin from 40 to 50 per cent; the color index is usually below 0 8; the leukocytes are from 20,000 to 40,000 to the cmm.; the lymphocytes predominate 60 to 70 per cent; monocytes are generally increased and embryonic red cells are common.

This disease tends toward recovery, though the majority of infants that have come to my notice with this disease have subsequently shown signs of constitutional inferiority either mental or physical.



BLOOD OF SPLENOMEDULLARY LEUKEMIA
(Anders and Boston, W. B Saunders Co)

Blood Diseases in Which the White Corpuscles Are Chiefly Affected

Leukopenia and *leukocytosis* are described in the Chapter on Blood Examinations, p. 1000.

Leukemia (Leukosis)

Leukemia is a disease characterized by an increase in the number of white corpuscles in the blood and is associated with hyperplasia of the bone marrow or the lymphatic tissue, or both (leukoblastic tissue). The two main types recognized are: (*a*) Myelocytic or myeloid (splenomedullary) and (*b*) lymphoid (lymphatic) leukemia

Myelocytic or Myeloid Leukemia* (Splenomedullary): *Symptoms and Diagnosis: This may be acute or chronic. In the chronic form, the onset *is insidious. The skin is somewhat pale and becomes paler as the disease progresses.* Epistaxis, gastrointestinal symptoms, sometimes hematemesis with *increasing loss of strength* are common symptoms The most prominent feature of this type is the enormous enlargement of the spleen accompanied by a definite blood picture. The leukocytes may increase to 100,000 or to 1,000,000 per cmm.; the average ratio between the white and red cells may be from 1 to 10. 1 to 5 or 1 to 1, instead of the normal 1 to 350 or 1 to 600. The polymorphonuclears usually show a reduction from 30 to 50 per cent. Small and large leukocytes, eosinophils and mast cells are increased. The myelocytes are increased to 30 per cent or to 50 per cent. As the disease progresses the red corpuscles and hemoglobin become markedly reduced.

Acute Myeloblastic Leukemia: This is characterized by its acute onset, ulcerations and hemorrhages in the mouth; the spleen and lymph glands are *enlarged, but* not quite as large as that found in myelocytic leukemia. The disease may be primary or it may be a terminal stage of *myelocytic leukemia.* The blood picture is that of a rapidly progressive anemia showing normoblasts and macrocytes with hyperchromia, or there may be microcytes with hypochromia. The white cell count *may at first* be *low*, but increases rapidly in a few days to 200,000 or 300,000; the predominating cells are *myeloblasts, though many premyelocytes* and some myelocytes are present. The blood platelet *count is low.*

Lymphocytic or Lymphatic Leukemia: *This* is characterized by hyperplasia of all the lymph glands. The spleen is but moderately enlarged. The liver is usually slightly enlarged. The blood shows a marked increase in the number of leukocytes, particularly of the lymphocytes, which number from 90 to 95 per cent of the entire white cell count

Acute Lymphatic Leukemia: This occurs in children and young adults as *a rapidly progressive fatal disease.* It is characterized by swelling of all the lymph glands in the neck, axillae, and other parts of the body. *Hemorrhages* from the mucous membranes into the serous sacs are common. The spleen is but slightly enlarged. The *blood* count shows an enormous increase in the number of leukocytes of which about 90 per *cent are lymphocytes* and *lymphoblasts.* The disease is rapidly fatal.

Leukemia Cutis: This is characterized by nodular masses in the skin which *disintegrate; hemorrhages* and discoloration of the skin and fever. The spleen and lymph glands are but little enlarged. The blood shows anemia with a great increase *in the leukocyte* count (one

million to two million per cmm), the greatest variety of which are lymphocytes

Chronic Lymphatic Leukemia: This is characterized by enlarged lymph glands in the neck, axillae and groins; moderately enlarged spleen and marked



Fig 2—Enormous enlargement of spleen

anemia, with an increase in the leukocyte count often numbering above 200,000, the greatest percentage of which are lymphocytes.

Atypical Leukemias

There are several varieties of atypical leukemia which may be described as follows:

Aleukemic Leukemia: This condition may be a stage of remission in leukemia or an atypical form of leukemia The spleen is enlarged or there may be enlarged lymph glands; the patient appears anemic, the leukocytes may not be increased in number but either lymphocytes or myelocytes are present in fairly large numbers.

Mixed Leukemia: This is in part myeloid and in part lymphoid. In nearly all cases of the ordinary splenomedullary leukemia a certain percentage of lymphocytes is present, and toward the end may be materially increased.

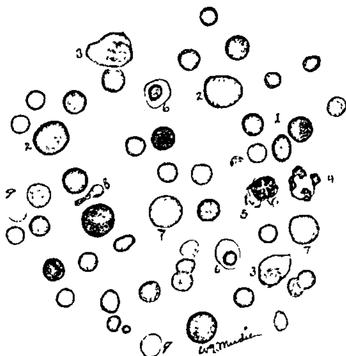
Chloroma: This is an atypical lymphoid leukemia presenting a leukemic blood picture and lymphatic tumors which are sarcomatous and possess a greenish color. It is commoner in children. Exophthalmos is frequent owing to tumor formation in the orbit. The tumor growths occur in the skull, the orbit, the cord, the long bones, and throughout the viscera. The lymph glands are affected, and changes occur in the spleen and the bone marrow. The typical picture of this disturbance may be present without the green tint of chloroma. The nature of the pigment is unknown

Leukanemia: This is a term invented by Leube to describe a condition showing features both of leukemia and severe anemia. The cases are now regarded as a myeloid leukemia with severe anemia. Glandular enlargement is usually present. The onset may be like the acute types of leukemia and the blood picture is either of the lymphoid or of the myeloid type

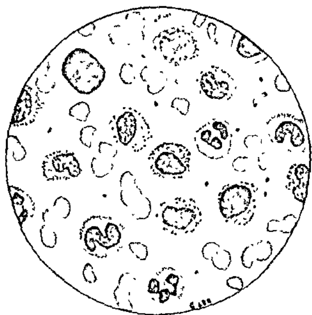
Cases with atypical blood changes, such as a very high percentage of eosinophils, or a condition with a very high proportion of plasma cells have also been reported.

In a few rare instances, a leukemic blood picture has been found without changes in the blood-making organs

Plasma Cell Leukemia: This type resembles lymphatic leukemia and runs a similar course though it differs in that quite a number of the abnormal cells



BLOOD OF LYMPHATIC LEUKEMIA
(Anders and Boston, W. B Saunders Co)



REPRESENTATIVE BLOOD SMEAR FROM A PATIENT WITH CHRONIC MYELOID LEUKEMIA

Note large number of mature and immature myeloid cells of all types (Hull, Wright & Eyles' "Medical Nursing" F. A. Davis Co., Philadelphia, Pa.)

in the blood and tissues are plasma cells. *In multiple myeloma the blood* picture is at times that of plasma cell leukemia.

Monocytic Leukemia: This closely resembles myeloblastic leukemia; the predominating cells are monocytes and may be identified as such by the use of the supravital stain of Sabin.

Basophilic Leukemia: This usually runs an acute course, the basophils may number from 50 to 60 per cent of the white cells present in the blood

Eosinophilic Leukemia: This usually runs a more or less chronic course, the blood may show from 40 to 50 per cent of the adult type of eosinophils.

Erythroleukemia: This is a rare type of leukemia which has the characteristics of both polycythemia and myelogenous leukemia The red cell count may be as high as seven or eight million and the white cell count may be from 200,000 to 500,000.

Leukemoid Reaction: This term is applied to a blood picture resembling chronic leukemia. The myelogenous type, in which the percentage of myelocytes is below 20, is found in malignancy affecting the bone marrow, in osteosclerosis and in certain infections. Leukemoid reactions of the lymphatic type are found at times in whooping cough, in infectious mononucleosis, in agranulocytosis and in other infections.

Subacute Leukemia: This occupies a position midway between the acute and the chronic forms. The onset is comparatively slow and may last several months. It is characterized by necrotic processes in the mouth or throat, moderate fever and progressive anemia. The *leukocytes are greatly increased and may* be of the lymphatic or myeloid type.

Pseudoleukemia: This is a blood disease resembling leukemia, to which Cohnheim has applied the name *pseudoleukemia. It is* doubtful whether this condition is a distinct entity as most cases of pseudoleukemia, after more careful study, have proven to be either Hodgkin's disease, generalized tuberculous lymphadenitis, *leukemia during its early stages* or during the state of remission, or a lymphosarcoma with metastasis only to the lymph glands. The general features of so-called pseudoleukemia are enlargement *of the lymph glands,* materially enlarged spleen and an absence of the typical leukemic blood picture. It is at times referred to as aleukemic leukemia



Fig 3—Lymphosarcoma (Jefferson Hospital).

Leukosarcoma

Leukosarcoma is a disease of the hematopoietic system characterized by the occurrence of tumor masses, the cells of which are either lymphoid, myeloid or both. It gives rise to widespread metastasis. The blood picture presents leukemic characteristics and the tumor masses are sarcomatous in structure.

Lymphosarcoma

This is characterized by the formation of malignant tumors in the lymph nodes which are spread by the lymphatics to the adjacent tissues; the spleen and bone marrow are seldom affected It usually occurs in the lymph glands of the neck, mediastinum, intestines, liver, tonsils, pleura, lungs, pericardium, brain and the bones In the early stages the blood presents only a secondary anemia with a moderate increase in the polymorphonuclear cells but late in the disease the lymphocytes are enormously increased This condition should be differentiated from adenitis, lymphoid leukemia, Hodgkin's disease and leukosarcoma

Agranulocytic Angina (Agranulocytosis, Granulocytopenia, Malignant Neutropenia)

This is a peculiar form of blood destruction occurring in the presence of severe infection. Agranulocytic angina is characterized by a severe ulcerative and gangrenous infection (often Vincent's) of the mouth, pharynx, larynx, or elsewhere. It is associated with high fever, prostration and a characteristic blood picture, *viz*, moderate reduction of red corpuscles, extreme leukopenia, *often* as low as 1000, marked reduction, or even total absence, of polymorphonuclear neutrophils. Lymphocytes and monocytes are abundant, often as high as 95 per cent; eosinophils and platelets are usually unaffected Granulocytopenia may be primary or secondary to local or general infection, to chemical poisons such as arsphenamine, bismuth, benzol, amidopyrine, sulfapyridine, sulfanilamide, and the barbiturates, also to exposures to x-rays and radium It may occur in aplastic anemia, leukemia and other blood diseases. This condition is exceedingly grave and recoveries are rare.

Infectious Mononucleosis (Glandular Fever)

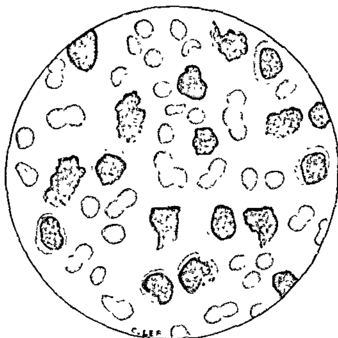
The blood picture is characterized by a normal red cell count and a leukocytosis of from 12,000 to 25,000, of which 50 to 85 per cent are lymphocytes, lymphoblasts and monocytes. The polymorphonuclear leukocytes may be reduced to from 50 to 85 per cent. Other features of this disease are acute cervical adenitis, pharyngitis, abdominal cramps, sweating and moderate rise in temperature. It usually occurs in children and young adults and as a rule terminates after two or three weeks in recovery. The *heterophilic antibody or agglutination test* is usually positive in high dilutions (SEE: pp. 203, 1059, 1064).

Hodgkin's Disease

(Lymphadenoma, Malignant Lymphoma, Lymphoblastoma)

Hodgkin's disease is a chronic granulomatous disease characterized by enlargement of the lymphoid tissue, progressive secondary anemia with enlargement of the spleen and liver.

Symptoms and Diagnosis: This disease is usually ushered in by painless enlargement of the lymph nodes, usually of the neck, axillary and inguinal regions They are bilateral, not tender to pressure and do not suppurate. The glands are freely movable beneath the skin and rarely become adherent. The heart usually becomes weak and pressure symptoms may occur in various parts of the body. Pressure against the cervical lymphatics will cause unilateral swelling of the face. Pressure upon the abdominal vessels will cause ascites, etc.



REPRESENTATIVE SMEAR FROM PATIENT WITH CHRONIC LYMPHATIC LEUKEMIA

Note large number of mature and degenerated lymphocytes (Hull, Wright & Eyl's "Medical Nursing," F. A. Davis Co., Philadelphia, Pa.)

SECTION 9

The Abdomen

The Blood Picture is that of secondary anemia and may at times show a moderate leukocytosis with an increase in the polymorphonuclear leukocytes and eosinophils, and at times also in the lymphocytes When in doubt a biopsy should be done for diagnostic purposes. The excised gland will present a characteristic microscopic appearance, *i. e.*, proliferation of the endothelial and reticular cells with the formation of uniform lymphoid cells; Dorothy Reed cells, giant cells, and lymphadenoma cells containing several nuclei. Eosinophils are always present and fibrosis of the gland is a common feature. In the later stages, the gland is usually hard and contains a greater abundance of fibroid tissues.

Osler and McCrea describe seven forms of Hodgkin's disease:

1. *Acute form;* in which the disease is ushered in with angina, simulating lymphatic leukemia, death occurring within a month or two.

2. *Localized form;* the enlargement may be localized to certain groups—those in the neck, the groin, the retroperitoneum or the thorax. The disease may be localized to one region for a year or more before it extends to other regions. The localized mediastinal group often presents a remarkable picture Pressure signs, such as pain, orthopnea, *dysphagia, hoarseness and, unless there* are other groups involved or enlargement of the spleen, the diagnosis of this group is often difficult.

3. *With relapsing pyrexia;* the relapsing pyrexia may occur in cases with involvement of the internal glands alone or more frequently with a general involvement of all the groups. The paroxysms of fever and remission may occupy several days and extend over a period of many months. During the fever the glands are enlarged, tender and hot. A case in the author's service at the Philadelphia General Hospital presented unusual features which led to a diagnosis first of typhoid fever, which was subsequently altered to miliary tuberculosis But on autopsy it was found that the retroperitoneal glands as well as the glands in the hili of the lungs were enlarged and showed characteristics of Hodgkin's disease

4. *Latent type;* the retroperitoneal glands or those of the hili of the lungs or of the hilus of the liver may become enlarged. Anemia, fever and weakness, and pressure symptoms usually occur.

5. *Splenomegalic form;* in which the spleen becomes very large, the lymph glands are not enlarged or but slightly so, and secondary anemia manifests itself. This condition should be differentiated from Banti's disease.

6. *Lymphogranulomatosis;* the skin lesions may be in the rare form of a true lymphogranulomatosis, or may show a variety of changes such as pruritus, urticaria, edema, petechiae and marked pigmentation.

7. *Lymphadenia ossium;* in this condition there are multiple bone tumors of the bone marrow and of the periosteum associated with enlargement of the lymph glands and spleen.

Prognosis: The course of the disease is usually chronic and is characterized by periods of remission. During exacerbation there may be irregular fever with signs of sepsis. The enlarged lymph glands and the tumor masses may for a time respond to x-ray exposures. They decrease in size rapidly. This treatment is effective for a time only. Eventually x-ray treatment as any other form of therapy becomes useless.

CHAPTER XX

Anatomy and Physical Examination of the Abdomen

The abdomen and its viscera are studied by inspection, palpation and percussion. Auscultation is of limited value in abdominal diagnosis Auscultatory percussion is employed to map out the outlines of various organs.

In order to study the abdomen and its viscera by physical exploration, familiarity with the anatomy of this portion of the body is necessary, as well as a thorough knowledge of the regional and relational anatomy of the organs it contains.

Anatomic Landmarks

To facilitate the study of the abdomen and its viscera, the abdomen, like the chest, is mapped out by anatomic landmarks *and defining lines into four* regions or into *nine* regions

By *the four-region division,* two lines are utilized by dividing the anterior abdominal wall. *One line passes vertically* through the umbilicus and separates the abdomen into two lateral halves. The other line passes horizontally through the umbilicus, dividing the abdomen into an upper and lower half, thus forming four quadrants, as follows:

1. An upper right quadrant.
2. An upper left quadrant.

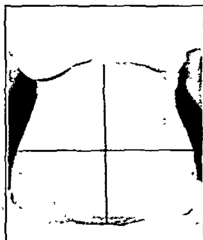


Fig. 1—Abdomen divided into four regions.

3. A lower right quadrant.
4. A lower left quadrant.

The contents of these quadrants, in addition to the peritoneum and omentum, are as follows:

Upper Right Quadrant

1. Right lobe of the liver
2. Gallbladder.
3. Hepatic flexure and part of the transverse colon.
4. *Portion of the pancreas.*
5. Pylorus
6. Right adrenal.
7. Right kidney.
8. Duodenum.

Lower Right Quadrant

1. Ascending colon.
2. Cecum
3. Appendix.
4. Right tube (in the female).
5. Right ovary (in the female).
6. Uterus when enlarged (in the female).
7. Bladder (when distended).
8. Small intestine.
9. Right ureter.
10. Right spermatic cord (in the male).

Upper Left Quadrant

1. Left lobe of liver.
2. Stomach.
3. Transverse colon.
4. Splenic flexure.
5. Pancreas.
6. Left adrenal.
7. Left kidney.
8. Spleen.

Lower Left Quadrant

1. Left tube (in the female).
2. Left ovary (in the female)
3. Uterus (in the female).
4. Bladder (when distended).
5. Descending colon.
6. Sigmoid flexure.
7. Left ureter.
8. Small intestine.
9. Left spermatic cord (in the male).

num, the pancreas, a section of the liver, the aorta, the solar plexus, and the celiac axis.

The *left hypochondriac region* contains the large end of the stomach, the spleen, the narrow extremity of the pancreas, the splenic flexure of the colon,

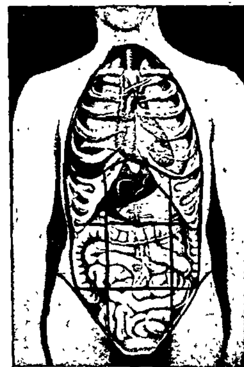


Fig. 4—The nine regions of the abdomen and their contents

the upper part of the left kidney and its suprarenal capsule, and sometimes part of the left lobe of the liver.

The *right lumbar region* contains the ascending colon, lower half of the right kidney, together with part of the duodenum and jejunum.

The *umbilical region* contains part of the omentum and mesentery, the transverse colon, the lower half of the duodenum, sections of the jejunum and ileum and the abdominal aorta.

The *left lumbar region* contains the descending colon, the lower half of the left kidney, and a part of the jejunum and ileum.

The *right iliac* or *inguinal region* contains the cecum, the appendix, McBurney's point, the lower end of the ileum, the right ureter, and the right spermatic cord in the male and the right ovary in the female.

The *hypogastric region* contains most of the ileum, the bladder (especially if distended) and the gravid uterus

The *left iliac* or *inguinal region* contains the sigmoid flexure of the colon, the left ureter, the left spermatic cord in the male and the left ovary in the female

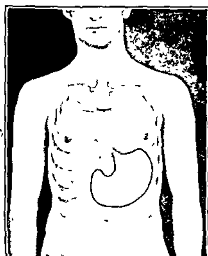


Fig 5—Position of stomach in relation to anterior abdominal wall and ribs

Topographic Anatomy of the Abdominal Viscera

The Stomach: This organ is situated in the upper portion of the abdomen, its fundus fitting into the dome of the left side of the diaphragm at the level of the fifth rib in the nipple line or below the heart apex. It is adjacent to the spleen, the lower border of the left lung,

To divide the abdomen into nine regions, two transverse circular lines are drawn around the body. One passes through the lowest point of the inferior costal border, which is usually the lower margin of the tenth costal cartilage; the other line passes over the anterior superior spines of the ilia. Thus, the abdominal cavity is divided into three segments Each of these is subdivided into three parts by two lines which are drawn *vertically along the outer borders of the* recti muscles; these lines correspond *practically to lines drawn vertically up*ward from the middle of Poupart's ligament *to join the midclavicular lines on* either side.

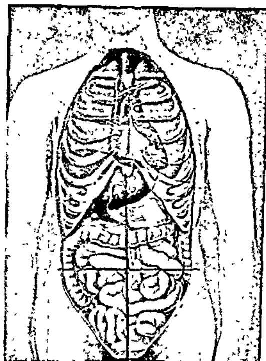


Fig 2—The four regions of the abdomen and their contents

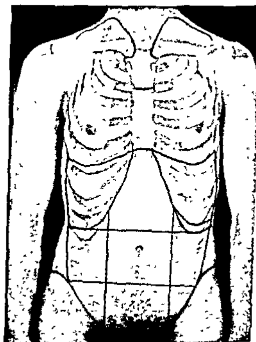


Fig 3—Division of the abdomen into nine regions by four lines

The two transverse circular lines are known as (*a*) the *subcostal line* (usually passing through the lower borders of the tenth ribs) and (*b*) the *bispinal line*, connecting both the anterior and *superior spines of the ilia.*

The nine regions thus mapped out upon the abdomen are

Right Side	Middle	Left Side
Right hypochondriac	Epigastric	Left hypochondriac
Right lumbar	Umbilical	Left lumbar
Right iliac or inguinal	Hypogastric	Left iliac or inguinal

The contents of these regions are as follows:

The *right hypochondriac region* contains the right lobe of the liver, the gallbladder, part of the duodenum, the he*patic flexure* of the colon, and part of the right kidney with its suprarenal gland

The *epigastric region* contains the pyloric end of the stomach, the duode-

The Liver (Hepar): The liver is the largest gland in the body It occupies nearly all of the right hypochondriac region and usually extends to the left hypochondriac region The *upper surface* of the right lobe is convex and fits

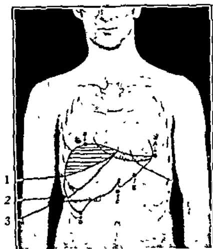


Fig 7—Anatomic position of the liver and gallbladder.

The horizontal shading indicates where lung covers the liver; vertical shading where heart overlaps the liver.

(1) Lower border of lung
(2) Lower lateral border of liver
(3) Gallbladder

into the dome of the diaphragm, extending upward as high as the fourth interspace, from which point the upper surface gradually declines so that in the epigastric region it is on a level with the base of the ensiform cartilage. The *lower boundaries* of the liver are·

Near the spine: At the eleventh rib.
Right midaxillary line: At the tenth rib
Right midclavicular line: At the lower margins of the ribs
In the median line· Midway between the ensiform and umbilicus.

Measurement: Upper surface of liver from A to B, 20 to 22 cm. (8 to 8¾ inches). Right lateral surface = convex line B to D, 15 to 17.5 cm. (6 to 6¾ inches). Lower edge of right lobe, D to C. Lower edge of left lobe, C to E and upwards to A. Anteroposterior diameter, at thickest portion it is 10 to 12.5 cm (4¾ inches) and at its thinnest portion 7.5 cm. (3 inches).

Weight: In the male the liver weighs 1.4 to 1.6 kg., in the female 1 2 to 1.4 kg.

The *anatomic outline of the upper boundary of the liver* should not be confused with the clinical boundaries or with the limits of absolute liver dullness. Clinically, the upper boundary of absolute liver dullness corresponds to the lower border of the right lung, *viz.:*

Anteriorly: Sixth rib.
Laterally: Eighth rib
Posteriorly. Tenth rib



Fig 8—Normal position of the spleen

The Gallbladder: The gallbladder is a serous sac which, in addition to other functions, acts as a reservoir for the storage of bile; it is situated at the undersurface of the right lobe of the

the heart, the left lobe of the liver, the left adrenal and kidney, and the aorta.

The *cardiac orifice* of the stomach lies to the left of the seventh sternochondral articulation, about four or five inches from the anterior surface of the body. The *pyloric orifice* is found to the right downward and forward, connecting the pylorus with the fundus. It forms the lower border of the stomach and, when the stomach is not distended, reaches to *about the level of the infracostal line* (tenth rib). Below, it is in close relation to the transverse colon.

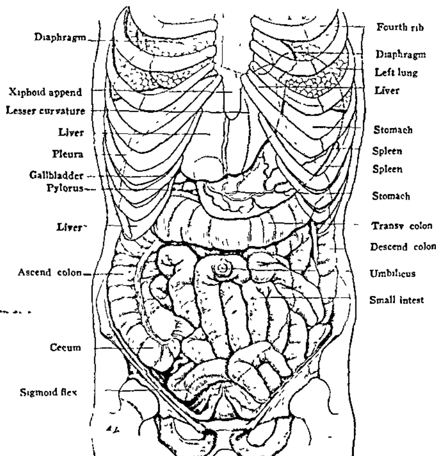


Fig 6—General topographic anatomy of the abdomen

of the midsternal line about two or three fingerbreadths below the ensiform cartilage, and directly behind the liver. It *is more superficial and has greater mobility* than the cardiac end.

The *lesser curvature* is slightly concave to the right and is situated to the left of the median line; it is in relation with the pancreas above and behind The *greater curvature* convexes gently

Only a small portion of the stomach is adjacent to the anterior abdominal wall in the epigastric region; another *superficial* portion of the stomach is found in Traube's semilunar space where gastric tympany can be elicited, this space is bounded above *by the lung,* and to the left by the spleen; *the right* boundary is formed by the left lobe of the liver.

tail (left extremity) is situated somewhat higher than the head, and is in contact with the spleen.

The Adrenal Glands: They are two in number, situated retroperitoneally, each imbedded in the perirenal fat above its respective kidney. The right adrenal measures 4 x 1 3 x .6 cm. (1½ x ½ x ¼ inch), and weighs 2 to 2.5 Gm. (30 to 40 gr). The left adrenal measures 4.5 x 2 x .6 cm (1¾ x ¾ x ¼ inch), and weighs 2 5 to 3 Gm. (40 to 45 gr.) (SEE: Fig 22, p 793).

The Kidneys: These two bean-shaped urinary excretors are situated on either side of the spinal column; each is about 10 cm. (4 inches) long, 6 5 cm (2½ inches) broad, and 2.5 cm (1 inch) thick. The kidneys are extraperitoneal organs supported by a mass of fat and resting upon the quadratus lumborum and psoas muscles, as well as upon the lumbar portions of the diaphragm. They lie on a level with the eleventh ribs and on a line continuous with the midclavicular line.

The relative positions of both kidneys vary to some extent.

RIGHT KIDNEY	LEFT KIDNEY
1. Is situated one-half inch lower than left.	1. Is situated one-half inch higher than right.
2 Upper border is in contact with the liver and reaches to the level of the eleventh dorsal spine; the duodenum and colon are anterior to it.	2. Upper border is in contact with the spleen and reaches to the eleventh rib; the colon lies anteriorly to it
3 Lower border posteriorly is 1¼ cm (one-half inch) below the third lumbar spine, or 2 5 cm (1 inch) above the iliac crest.	3 Lower border posteriorly is on level with the third lumbar spine, or 3 75 cm (1½ inches) above the iliac crest.
4. Anteriorly the lower border extends to about 2 5 cm (1 inch) above the horizontal umbilical line	4 Anteriorly the lower border extends to about 3 75 cm. (1½ inches) above the umbilical line.
5 Shorter and thicker than left.	5 Longer and thinner than the right kidney.

The Intestines: The *small intestine* occupies nearly the entire central portion of the abdomen, excepting the duodenum; it is freely movable and the various divisions are continuous, so that it is practically impossible to determine by palpation where the ileum ends and the jejunum begins. The jejunum is usually found in the upper part of the abdomen, and toward the left, *i. e.*, left lumbar, left iliac, and left half of the umbilical region, while the coils of the ileum occupy a lower position on the corresponding right side.

The *large intestine* is more fixed than is the small intestine. The cecum is located in the right iliac region, resting on the right psoas muscle and corresponding to the center of a line drawn from the anterior superior spine of the ilium to the symphysis pubis. The ileocecal valve is on a level with the iliac line, about three inches internal to the anterior superior spine.

The *vermiform appendix* arises from the inner and posterior aspect of the cecum near the ileocecal valve; its base corresponds to a point which is the center of a line drawn from the anterior superior spine of the ilium to the umbilicus, and corresponds at that point to the right edge of the rectus muscle; it is about two inches from the right anterior superior spine of the ilium (McBurney's point).

liver, its fundus extending downward. The fundus is ordinarily located at the outer border of the right rectus muscle, on a level with the inner edge of the ninth costal cartilage.

The Spleen (Lien): The spleen is a soft, vascular, oval-shaped organ, meas-

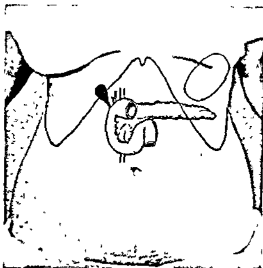


Fig. 9—Relation of pancreas to gallbladder, duodenum, left costal angle and spleen

uring about 12 cm. (4½ inches) in *length and 7 cm. (3 inches) in width*, and 3 to 4 cm (1¾ to 1½ inches) in thickness, and weighs about 200 Gm (6½ ounces). The spleen is situated in the left hypochondriac region between the ninth and eleventh ribs, its long axis being parallel with these ribs. Its outer surface is convex and is in relation to the diaphragm, while the inner *surface is* concave. *Posteriorly* it is in relation to the suprarenal capsule and upper part of the left kidney. *Anteriorly*, it is in relation to the outer portion of the cardiac end of the stomach and the splenic flexure. The *lower two-thirds* of the spleen are in contact with the ribs; the *upper one-third is separated from them by the* diaphragm and lung. The hilum of the spleen can be felt only when this organ is greatly enlarged.

The Pancreas: The *pancreas is a* long flattened gland measuring from 12.5 to 15 cm. (5 to 6 inches) in length, about 5 cm. (2 inches) in breadth, and 2.5 cm (1 inch) in thickness. It weighs between 60 and 110 Gm It is deeply situated in the epigastrium, extending from the right to the left hypochondrium and

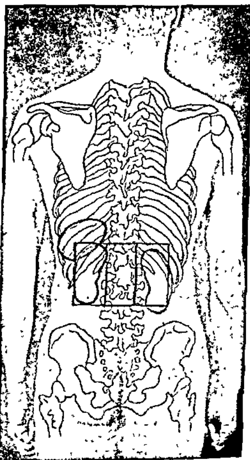


Fig 10—Relation of kidneys to spleen, to spinous processes and ribs.

lying behind the stomach at a level with the first lumbar vertebra. The head of the pancreas (right extremity) extends to the right of the median line, a little *above the subcostal line*, and is embraced by the curvature of the duodenum. Its

is no ascending colon at all, and the cecum lies directly against the edge of the liver, then it is within the border line of a left-sided position. This may be recognized when the cecum is so far displaced to the left that the large and small intestines no longer decussate

The *appendix* will be displaced in any of the congenital conditions mentioned above. In enteroptosis it may lie in the true pelvis When the cecum is abnormally high or when the ascending colon is abnormally short, the appendix may be found high up in front of the right kidney, at the edge of the liver, or even under the liver close to the gallbladder. When the large intestine is displaced to the left, the appendix usually lies in the umbilical region or even to the left of it, while in complete transposition, the appendix will be on the left side of the pelvic cavity

Acquired Displacements: These are grouped together under the term of enteroptosis, even if the displacement concerns only one viscus. *Glénard's disease* (splanchnoptosis, enteroptosis) may be congenital or acquired. The mesenteric and peritoneal attachments of the stomach, intestines, transverse colon, liver, spleen and kidneys are stretched so that these structures occupy an abnormally low position in the abdomen. The acquired type is generally due to a lowering of the intraäbdominal pressure caused by weakening of the abdominal, pelvic and to some extent the spinal muscles.

"Floating kidney," due to lack of support, occurs more frequently on the right; *movable liver and spleen* occur rarely; a movable liver is due to general relaxation of the suspensory ligaments, while a movable spleen is caused by some pathologic enlargement of that organ or a lengthening of its pedicle A movable spleen is easily recognized by its sharp anterior border, the notch, and by the fact that splenic dullness is absent from its normal situation

Palpation is of great value in a diagnosis of acquired ptosis, but an x-ray examination will be of greater service, and should be called to the aid of physical examination whenever possible.

Inspection of the Abdomen and Its Viscera

Inspection of the abdomen is usually performed with the patient in the recumbent posture, though at times, for special reasons (to note a pendulous condition, hernia, engorged veins, or the shifting of tumors or other masses) the erect and sitting postures are employed.

Technic: The patient lies flat upon his back, allowing the dependent parts of the body to rest naturally upon the bed or the examining table. The entire abdomen must be exposed to the examiner's view, this is best accomplished in sensitive females by covering the body with a sheet or blanket; under this cover the nightdress is gently drawn up as far as the lower part of the sternum, then the upper covering (sheet or blanket) is folded downward to the pubis, exposing as little as possible of the mons veneris. The examiner takes a position at that side of the patient which allows a good light to fall directly upon the part under examination, and at the same time permits him to view the abdomen from various angles. It is at times necessary to bring the eyes down to the level of the patient's abdomen so as to inspect for movements and pulsations

The Bladder: Under normal conditions the bladder does not extend above the pubic arch, but when greatly distended, *it may rise to the level of the* superior spines of the ilia.

The Abdominal Aorta: The abdominal aorta begins *at the twelfth dorsal* vertebra, thence it passes down the left side of the spinal column to the fourth lumbar vertebra at which point it bifurcates into the right and left iliac arteries

Displacement of the Abdominal Viscera

When the abdomen is examined for any pathological condition, it is customary to assume that the viscera occupy their normal positions. It is, however, quite possible that one or several of them may be displaced to a greater or lesser degree, and the success of abdominal diagnosis may often rest upon a thorough appreciation of this possibility

Congenital Displacements: The commonest variety is *situs inversus viscerum. This is detected with compara*tive ease if the thoracic viscera are similarly displaced, but if the displacement exists only in the abdomen it is much more likely to be overlooked, though palpation and percussion may reveal those rare cases where the positions of liver and spleen are reversed. Under such circumstances, the stomach will be upon the right side, and the appendix in the left iliac fossa, the findings of physical examination can readily be confirmed by x-rays.

Displacements of the intestine alone are much more common; the following varieties have been distinguished by de Quervain·[1]

"1. The large intestine lies in its whole extent, *behind the small intestine,* because of the failure of the umbilical loop to rotate (retroposition). The mesentery may either be free or may contract adhesions with the posterior abdominal wall

"2 The entire large intestine lies on the *left side of the abdomen,* because, although the umbilical loop has rotated in the right direction, it has failed to do so completely, *i. e.,* to the extent of permitting decussation of the small and large intestine (*sinistro-position*). The mesentery may either be free or may have contracted secondary adhesions. In the first case both small and large intestine are connected with a free common mesentery, the so-called *mesenterium commune.*

"3. The entire large intestine is in the *right half of the abdomen* because the umbilical loop has incompletely rotated in the wrong direction (*dextro-position*). The condition of the mesentery is the same as in 2.

"4. There has been complete decussation of the small and large intestine, but in a *reversed position,* because, though the umbilical loop has revolved completely, the direction has been wrong (*situs inversus abdominalis partialis inferior*)."

These are the extreme varieties, but a much more frequent abnormality is one which may be regarded as an intermediate form between the normal position and the left-sided position of the large intestine, with free mesentery. Here the cecum and the ascending colon possess a free mesentery, which merges with that of the lowest coil of the small intestine. At the same time the ascending colon is frequently shortened, so that the cecum is abnormally high. If there

[1] de Quervain: Clinical Surgical Diagnosis, translated by J. Snowman, J Bale, Sons and Danielsson, London, 1921.

(*b*) Addison's disease; generalized dirty-brown color, with a darker area around the waistline.

(*c*) Syphilitic discoloration, copper colored

(*d*) Albinism; white areas, irregularly situated.

(*e*) Linea albicantes; white lines due to previous overstretching of the skin, as after pregnancy, ascites and loss of fat.

(*f*) Linea nigra; a dark line stretching from the umbilicus to symphysis pubis, seen in pregnancy and chronic abdominal enlargement.

(*g*) Bluish or purplish striae upon the abdomen and upper thighs are found in Cushing's syndrome

(*h*) Pernicious anemia; pale, lemon yellow or straw color.

(*i*) Hemochromatosis; dark brown to leaden or bluish black color.

Rashes: (*a*) In patients suffering *from typhoid fever rose-colored spots* or small lenticular macules occur in small groups on the eighth day of the disease and disappear after a few days, then recur in successive crops They are usually found over the lower chest and upper abdomen, disappearing on pressure and reappearing when the pressure is removed

(*b*) Copper-colored, scaly, somewhat circular spots are often seen in secondary syphilis

(*c*) Raised white areas surrounded by reddened areas, which are evanescent and itchy, are indicative of urticaria

(*d*) Lesions covered with white, "mother-of-pearl" scales are indicative of psoriasis.

(*e*) Groups of vesicles arising from an erythematous base that itch or burn are indicative of herpes zoster.

(*f*) Scratch marks may be found in jaundice, pediculosis, scabies and other conditions that cause intense itching.

(*g*) A macular or maculopapular rash in which the lesions are oval, scaly, bright rose, and later present a yellow center with rosy edges, are usually due to pityriasis rosae.

(*h*) Brown spots of varying sizes, somewhat raised and covered with fine furfuraceous scales, are due to tinea vesicolor.

(*i*) Various skin lesions found over other parts of the body also occur on the abdomen.

Abdominal *scars* are a result of healed lesions, traumatism to the abdominal wall, or the healing of surgical incisions A longitudinal scar in midabdomen above the umbilicus may indicate a previous operation upon the stomach, pancreas or intestines; in the right upper quadrant a liver or gallbladder operation, and below and to the right of the umbilicus an appendiceal operation A longitudinal scar in midabdomen below the umbilicus may be the result of an exploratory incision, an omental or bowel operation In old men it may be a result of prostatectomy; while in women such a scar may indicate the occurrence of a previous pelvic operation or a cesarean section A long scar in either or both inguinal regions may be the result of a hernia operation and a scar in the kidney region may indicate that there has been some renal operation

General Nutrition: A large abdomen in a fat person is found in general obesity of the pituitary type or it may not denote an abnormal condition, but a large abdomen with taut and glistening skin indicates ascites, peritonitis, or chronic bowel distention. In women, in addition to the conditions mentioned, such a appear-

The object of inspection is to note: (I) The skin of the abdomen; its color, the presence of rashes or scars, and the general state of nutrition; (II) the enlargement of superficial veins; (III) pulsations and enlarged arteries; (IV) the condition of the umbilicus; (V) peristalsis; (VI) respiratory movements; (VII) size, shape and symmetry of the abdomen.



Fig. 11—Inspection of abdomen for peristaltic movements and local bulgings.

The Normal Abdomen

The *skin* is usually of the same color as that of the rest of the body, though the lower portion is somewhat darker than the upper, and is usually covered with coarse hair. In brunettes the linea nigra (a dark line at the junction of both recti muscles and running parallel with them from the umbilicus to the symphysis pubis) is fairly prominent. Rashes are absent and scars occur only as a result of a previous surgical operation or an accidental wound. The general nutrition is in keeping with the rest of the body.

Superficial veins are not visible, though at times one or two slightly distended veins can be seen, running a short distance up the abdomen from either or both inguinal regions.

Pulsations are not evident except during excitement or after exercise when epigastric pulsations may be noticed.

The *umbilicus* is depressed and the skin around it is folded inward.

Peristalsis is usually not apparent unless the examination is made shortly after a full meal, following the taking of a cathartic, or when the skin of the abdomen is irritated by manipulation, or other cause.

Respiratory movements are visible in men and young children but as a rule are not very noticeable in women.

The *size and shape* of the abdomen are in keeping with the rest of the body, large in the obese, gently convexed and oval in the well nourished, flattened in *thin or undernourished, even though* healthy, adults In children the abdomen is globular. It is usually symmetrical on both sides, somewhat fuller above the umbilicus than below it. In males during early adult life there is generally a depression in the epigastric region. The dimensions of the abdomen vary within wide limits, depending upon the amount of subcutaneous tissue and omental fat. In women the lower portion of the abdomen or the pelvic region is broader than in men.

The Pathologic Abdomen

The Skin: ***Color:*** Discoloration and pigmentation of the skin over the abdomen may be general, in keeping with discoloration and pigmentation of the rest of the body, or it may occur as a local condition.

Generalized discoloration and pigmentation is observed in:

(*a*) Jaundice; deep yellow, orange tinge or lemon yellow.

sating empyema; in the iliac regions, by a lesion of the heart (aortic regurgitation); in the inguinal regions, by inflammatory lesions and by partial obstruction of the abdominal aorta.

Partial obstruction of the abdominal aorta or iliac arteries (rare) may cause enlarged and visible arteries in the epigastrium



Fig 13—Omphalocele (Umbilical hernia)

Condition of the Umbilicus: In fat subjects the umbilicus is deeply retracted; it protrudes in umbilical hernia (omphalocele), massive ascites and portal obstruction, and is flattened in the presence of moderate abdominal effusions, tumors and pregnancy. The umbilicus may likewise be inflamed, eczematous, and, in rare cases, exude a foul-smelling discharge.

Peristalsis: Visible peristaltic movements are an indication of hyperactivity of the bowel or stomach; this may be seen in colitis, in partial intestinal obstruction, and in complete obstruction above the obstructing point. Reversed peristalsis is often noted in intestinal and pyloric obstruction.

Respiratory Movements: Respiratory abdominal movements are very much in evidence in normal men and young children, but much less so in women.

Pathologically increased abdominal respiratory movements are caused by some diseases of the chest, which do not permit chest expansion, *i. e.,* inflammatory condition of the lung and pleura, consolidation of the lung, large pleural effusions (fluid or air), and broken ribs which may be the cause of pleuritis and muscular rigidity, also by chronic emphysema, asthma and pulmonary edema

Diminished or *absent* respiratory movements may be caused by large tumors in the abdomen, upward pressure of the diaphragm by enlarged abdominal viscera, painful condition of the abdominal muscles, inflammatory condition of the peritoneum, or by ascites.

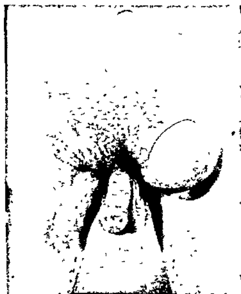


Fig 14—Femoral hernia.

Size, Shape and Symmetry: *General enlargement* of the abdomen, if not

ance of the abdomen may be due to pregnancy, ovarian cyst or other tumors.

An enlarged abdomen not due to fat, the skin of which is not glistening, may be caused by edema of the abdominal wall, by an enlarged liver or spleen or by enlargement of both organs and by distention of the bowels, by large abdominal tumors, cysts, distended bladder or by peritonitis and ascites.



Fig 12—Enlarged superficial abdominal veins (Phila General Hospital)

Children and young adults, if idiots, cretins, or sufferers from uncinariasis, usually present large abdomens.

Enlarged Superficial Veins: Enlarged superficial veins usually indicate obstruction to the return circulation.

Caput medusa consists of a number of enlarged veins radiating from the umbilicus; this is due to dilatation of the cutaneous veins and is indicative of portal obstruction. It may rarely be found in the newborn, and is seen also in atrophic cirrhosis of the liver and in abdominal tumors.

General enlargement of the abdominal veins may indicate obstruction to the return circulation caused by an enlarged liver, by tumor or abscess of the liver, by syphilis of the liver or omentum (gumma), by chronic distention of the stomach or other viscera and by tumors of the mediastinum (SEE: p. 384c).

When a distended vein is emptied by pressure, its mode of refilling should be noted. If the vein fills from above downward it is generally due to compression of the superior vena cava, for the blood from this vessel forms a collateral circulation by way of the azygos veins communicating with its many tributaries If the vein fills from below upward, it is indicative of obstruction of the portal vein and inferior vena cava Veins distended only in the pubic region are usually due to some obstruction below the liver

Pulsations and Enlarged Superficial Abdominal Arteries: Epigastric pulsation may be caused by a dilated right heart, a dynamic aorta, an aneurysm of the celiac axis or of the abdominal aorta A tumor of the stomach. of the pancreas or of a portion of the omentum overlying the abdominal aorta may cause transmitted pulsation, as will also a pulsating liver.

Pulsations in the upper abdomen may indicate a tumor overlying the aorta, aortic aneurysm, or unusual thinness of the abdominal wall which is in close contact with the aorta. Abdominal pulsations are often seen in neurasthenic individuals

Pulsations in the lower abdomen may be caused by an enlarged pulsating liver (tricuspid regurgitation), or by a pul-

tonitis, tuberculous peritonitis (chronic), Acute intestinal obstruction, chronic lead poisoning, mesenteric thrombosis, Hirschprung's disease, rupture of an abdominal aneurysm, rupture of the intestine, or stomach, ileus, acute and chronic enterocolitis, the various types of colitis, amebic or bacillary dysentery, food poisoning, periarteritis nodosa, ab-

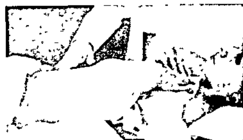


Fig. 17—Technic for palpating in appendiceal region for muscle rigidity and tenderness

dominal neuralgia, tabes dorsalis, arsenic and mercury poisoning, retroperitoneal malignancy, Asiatic cholera, the early stages of meningitis, or possibly a reflex from some chest, spine or cord condition.

Local tenderness, if elicited over the *right lower quadrant,* may be a sign of appendicitis, carcinoma of the colon, regional ileitis, acute diverticulitis, fecal impaction, spastic colon, psoas abscess, incarcerated hernia, or obstruction of the ureter by the passage of a stone It may also be a reflex from a tuberculous process of the ileum, or some inflammatory condition of the spermatic cord In women, it may be caused by an inflammatory condition of the Fallopian tube or of the ovary. Certain chest diseases and inflammatory conditions of the diaphragm may cause reflex tenderness in this area

Tenderness in the *right upper quadrant* is produced by an inflamed gallbladder, or an inflammatory condition of the liver, such as an abscess, hydatid cyst, gumma, malignant disease, acute cholangitis, diaphragmatic abscess or pleurisy, right-sided pleurisy or malignant disease of the chest "De Mussy's point" is a tender point corresponding to a small area intersected by the midclavicular line and a horizontal line continuous with the tenth rib The presence of the tender area indicates diaphragmatic inflammation or gallbladder disease.

Epigastric tenderness usually indicates an acute inflammatory condition; it is found in ulcer and cancer of the stomach, gastralgia, ulcer or cancer of the duodenum, in acute pancreatitis, subphrenic abscess, and also in myocarditis, coronary sclerosis, mediastinitis, tumors, aneurysm of the aorta, aortitis, or erosion of a vertebra.

Tenderness in the *left upper quadrant* may be caused by an inflammatory condition of the kidney, spleen, suprarenal capsule, or the cardiac end of the stomach; likewise by a local inflammation of the splenic flexure and omentum Left-sided pleurisy, diaphragmatic hernia, diaphragmatitis, aneurysm of the thoracic aorta, and malignant disease of the lung may reflexly produce left-sided upper abdominal tenderness

Tenderness in the *left lower quadrant* may be due to obstruction to the left ureter, incarcerated left hernia, malignant disease of the sigmoid, spastic colon or orchitis. In women, disease of the left ovary and Fallopian tube should be borne in mind as possible causative factors.

Tenderness above the *symphysis pubis* may be the result of an inflammatory condition of the urinary bladder or disease of the symphysis pubis; in the

Rigidity in the right upper quadrant may be due to cholecystitis; cholelithiasis; abscess, gumma, or general enlargement or inflammation of the liver; abscess of the right kidney, or some other inflammatory condition of the kidney structure; hypernephroma; diaphragmatic inflammations, abscess or cyst; retroperitoneal sarcoma and inflammatory conditions of the adrenal body.

Rigidity in the left lower quadrant may result from an inflammatory condition of the left ovary or tube, or a pathologic condition of the sigmoid, *e. g.*, carcinoma, local peritonitis, or from diverticulitis or strangulated or incarcerated hernia or undescended testicle.

Rigidity in the left upper quadrant usually indicates disease of the spleen, left kidney, retroperitoneal sarcoma, hypernephroma, subdiaphragmatic abscess, inflammatory conditions of the adrenals diaphragmatic pleurisy, herpes zoster, and occasionally occurs reflexly from inflammation of the tail of the pancreas or of the bile ducts, and, at times, in basal pneumonia.

Rigidity of the upper midabdomen may be caused by gastric carcinoma or ulcer, by disease of the pancreas, aortic aneurysm, periarteritis nodosa, retroperitoneal malignancy or by disease of a vertebra.

Rigidity of the entire abdomen may be caused by general peritonitis, intussusception or acute obstruction of the bowel from any cause, Asiatic cholera, meningitis, lead colic, or any other condition causing spasm of the abdominal muscles, i.e., abdominal adhesions, distention of the bowels, spinal injury, etc. Apparent superficial rigidity is sometimes found in cases of pneumonia, particularly in children and in spinal nerve injury.

II. Tenderness: Abdominal tenderness is usually an indication of some inflammatory condition of the peritoneum as a whole, of a portion of the peritoneum overlying an inflamed viscus or of inflammation or injury of the abdominal wall or its innervation.

Technic for Eliciting Tenderness: With the patient in a supine position and being careful to eliminate all avoidable muscular rigidity, the examiner gently touches the various portions of the abdomen with his warm hand. In order to elicit tenderness more precisely, he should use the palmar surface of the first four fingers

Palpation should at first be very light, gradually increased in force as the case permits If the pressure of the hand causes severe pain, it is best to outline the painful area by light palpation, starting at a point far away from the seat of acute pain, and gradually coming towards it. The point at which pain is first felt by the patient is marked as the outer limit of the painful area. In this way, as a rule, the diseased portion can be approached from all angles; whenever pain or tenderness is felt by the patient, rigidity—either marked or slight, as the case may be—can be perceived by the examiner.

General tenderness over the entire abdomen can be recognized both by rigidity of the abdominal muscles, and by the pain elicited by touching the various portions of the abdominal surface

Occasionally there may be superficial or skin tenderness elicited by light touch, and not felt by deep palpation. This is usually due to affection of the nerves supplying the skin or to local skin irritation.

Tenderness over the entire abdomen may denote the presence of acute peri-

tonitis, tuberculous peritonitis (chronic), Acute intestinal obstruction, chronic lead poisoning, mesenteric thrombosis, Hirschprung's disease, rupture of an abdominal aneurysm, rupture of the intestine, or stomach, ileus, acute and chronic enterocolitis, the various types of colitis, amebic or bacillary dysentery, food poisoning, periarteritis nodosa, ab-

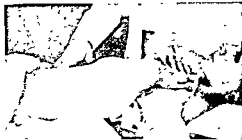


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Local tenderness, if elicited over the *right lower quadrant,* may be a sign of appendicitis, carcinoma of the colon, regional ileitis, acute diverticulitis, fecal impaction, spastic colon, psoas abscess, incarcerated hernia, or obstruction of the ureter by the passage of a stone. It may also be a reflex from a tuberculous process of the ileum, or some inflammatory condition of the spermatic cord. In women, it may be caused by an inflammatory condition of the Fallopian tube or of the ovary. Certain chest diseases and inflammatory conditions of the diaphragm may cause reflex tenderness in this area.

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Tenderness above the *symphysis pubis* may be the result of an inflammatory condition of the urinary bladder or disease of the symphysis pubis; in the

ably indicates a distended gallbladder, hydatid cyst, abscess, gumma or malignant tumor of the liver, a cystic or otherwise enlarged kidney or hypernephroma.

Small nodular or bosselated masses on the liver surface are found in atrophic cirrhosis of the liver, malignancy, syphilis, Hodgkin's disease and hepatic tuberculosis. If a tumor mass is connected with the liver, spleen or kidney, when these structures are in contact with the diaphragm, a downward displacement will be felt during inspiration Tumor masses that are soft and yielding may be caused by dilatation of the intestines or stomach Tumors in the lower portion of the abdomen may result from ovarian cyst, uterine fibroid, ectopic pregnancy, tuberculous peritonitis, Hodgkin's disease and fecal masses or concretions.

Diagnosis: In the diagnosis of abdominal tumors, Butler[1] offers the following suggestions:

"*Points to be Observed:* If one is satisfied of the presence of a tumor, the following points remain for determination:

"Is it intraäbdominal or extraäbdominal? *Is it freely movable and does it* move with respiration? What is its size, shape, consistence, the nature of its surface? Does it fluctuate? In what region of the abdomen does it lie? From what organ, if any, does it spring?

"1 If situated in the abdominal wall, it is usually possible to gather up, either in one hand or between both, that portion of the abdominal wall overlying the tumor, when the latter can be distinctly felt to lie in the grasp of the hand. An intraabdominal growth, on the contrary, *cannot thus be elevated and seized,* the abdominal wall slipping easily over it unless it has contracted firm parietal adhesions.

"2. The mobility of the tumor should be tested by moving it in various directions, observing the extent of movement and the line in which it is most readily pushed, *e. g.,* floating kidney, which is most easily carried upward and backward.



Fig 21—Enlargement of abdomen due to large liver.

"If, when the hand is laid upon the tumor, the latter is found to move up and down with each respiration, it may be inferred that it springs from organs in close relation with the diaphragm, *i. e.,* liver, spleen, and, to a less extent, the *kidney. This is a sign which* possesses considerable diagnostic value, but it must be remembered that the tumor may have contracted adhesions in such a manner as to produce the same effect On the other hand, tumors which would ordinarily move with respiration may be hindered from so doing by interference with the contraction of the diaphragm consequent upon pleurisy, emphysema, or a greatly *enlarged liver or spleen.*

"The tumors which are *readily movable by palpation,* and which descend when the patient is in the erect position,

[1] Butler, G R.: The Diagnostics of Internal Medicine, D. Appleton & Co.

are floating liver, spleen and kidney; tumor of the stomach (especially pyloric) or intestine; fecal masses or concretions, and gallstones. *Slightly movable* are tumors of the gallbladder and omentum above; of the uterus and ovaries below. *Immovable* are tumors of the pancreas, aneurysm of the abdominal aorta, abscess or inflammation due to disease of the appendix, tumor of bone or abscess resulting from caries, and enlarged retroperitoneal glands or abscess Tumors of the stomach or intestine may change position with the peristaltic movements.

"3 Note also its size, approximately or by measurement; its shape, round, ovoid, or irregular; its surface, whether smooth or nodular; and its consistency—soft, doughy, and indentable (fecal mass), moderately hard or stony Can fluctuation be obtained, *i. e.*, is it of a cystic nature, with fluid or semifluid contents (hydronephrosis or pyonephrosis, ovarian cystoma, distended bladder, hydatid cyst, pregnant uterus, ectopic gestation, or encysted abscess)? If fluctuation is present, test for the 'hydatid thrill,' by placing three fingers over the fluctuating mass and percussing strongly upon the middle one of the three, letting the plexor or striking finger rest at the end of each stroke, when, if the thrill is elicited, it will be perceived by the two lateral fingers.

"4. Observe carefully in what part or region of the abdomen the swelling or tumor lies.

"5. Determine as accurately as possible whether it is entirely of abdominal origin, or whether it springs from the pelvis Careful deep palpation, just above the brim of the pelvis, together with a rectal or vaginal examination, will usually determine this point, but cases occur in which errors are quite possible, *e.g.*, an abscess of the ovary rising out of the pelvis, sufficiently high to be diagnosed as an appendiceal abscess.

"A decision as to the particular organ or structure from which a tumor springs, or a diagnosis of the nature and seat of the disease causing local swelling or bulging in various parts of the abdomen, depends not only upon the location and character of the tumor or swelling, but also, and often to a large extent, upon the history of the case and the results of chemical and microscopical examinations of the sputum, gastric contents, blood, urine, or feces, and the x-ray findings

"*Indications Derived from the Situation of Abdominal Swelling or Tumors:* For the sake of clinical convenience in describing the significance of swellings or tumors according to the part of the abdomen in which they are found, one may recognize seven areas or regions, each named, with two exceptions (pelvic and umbilical), after the most important organ or part underlying it. These areas—the boundaries of which necessarily overlap to some extent—are in the median line, gastric, umbilical and pelvic; to the right, the hepatic and appendiceal; to the left, the splenic and sigmoid. Furthermore, as certain bulgings or tumors may occupy almost any point in the abdominal cavity, it is practicable to form, according to their distribution but with some necessary repetition, eight groups of palpable abdominal lesions. It is helpful from a diagnostic viewpoint to have in mind the possible findings when palpating and percussing special regions or areas of the abdomen. It is to be remembered that a tumor or an enlarged organ in one of these areas may grow to such dimensions that it underlies several of these areas, or indeed, may occupy nearly the entire abdominal cavity—*e g.*,

placed or in very thin individuals). All other abdominal organs cannot, as a rule, be outlined by palpation alone.

Technic for Palpating Abdominal Organs: *Liver:* The patient lies supine, avoiding all muscular rigidity. In order to have the abdominal muscles more flaccid, the thighs should be somewhat drawn up, the shoulders raised and supported by a pillow; the patient should be instructed to breathe regularly, preferably through the mouth The examiner places one hand over the patient's right flank, so as to support it, the other hand being placed, fingers pointing upward, on the anterior aspect of the abdomen at a point some distance below the liver's normal position The hand which is placed on the anterior abdominal wall is moved slightly upward, while the patient is instructed to take a deep breath. The descent of the edge of the liver can be felt by the palpating hand during the patient's inspiration and its ascent during expiration.

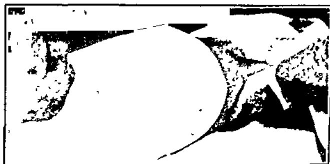


Fig 22—Technic for palpating liver.

Kidney: Under normal conditions, the kidney is not palpable unless the individual is very thin, but if displaced, it can readily be felt when the two hands are held in the position described for liver palpation. The kidney is recognized as such by its hilum and by its mobility. When displaced, it is not, as a rule, influenced by respiration.

Spleen: Normally the spleen cannot be located by touch, but when enlarged, its palpability depends upon its size. A moderately enlarged spleen, such as is found in typhoid fever, can be felt in the left hypochondriac region immediately below the left costal margin. The examiner placing his hand below the costal margin, the patient is instructed to take a deep breath while the examiner moves his palpating hand upward. At the height of inspiration, the hand and spleen usually meet. A moderately enlarged spleen is recognized as such by its hilum A very large spleen may be felt in the left half of the abdomen as a hard, freely movable mass anterior to the bowel.



Fig. 23—Palpating for spleen or kidney.

VI. **The Size of Abdominal Organs:** The abdominal organs cannot be definitely outlined by palpation alone;

percussion is usually required as an aid. Even when enlarged, only the exposed portions of the liver, spleen and kidney can be *outlined by palpation*, while that part of the liver, kidneys, and spleen situated within the thorax must be demonstrated by percussion The stomach may be approximately outlined by palpa-



Fig 24—Mediate percussion of abdomen, locating lower edge of stomach

tion only when it is greatly distended, and not very accurately at that; the pancreas and other deeply situated abdominal organs (except the uterus), can never be palpated with any degree of accuracy In order to outline the size of an enlarged liver, the technic employed is similar to that used for locating the other abdominal organs; in addition to which, the hand may be made to conform gently to its outlines, so that its consistency, size and the shape of its edge can thus be determined. The spleen is palpated in the same manner as is the liver Its size, consistency and shape may be determined with the finger tips, always being careful to have the patient breathe deeply, so as to cause as much mobility as possible A kidney, when displaced and movable, can be grasped between the hands and moved a considerable distance from its original location, or it may be pushed up to its normal position. A very large kidney should be palpated for its consistency, in order to determine if it be *cystic, hydronephrotic or the seat of an abscess.* In the case of abscess, the kidney is felt as a soft boggy, often fluctuating, mass. *If the enlargement is due to amyloid* disease, or any other condition affecting the interstitial structure of the kidney, it can be felt as a hard, roughly bean-shaped organ.

Percussion of the Abdomen and Its Viscera

Though percussion of the abdomen is secondary in importance to palpation, it is useful in confirming inspected and palpated signs and in demonstrating the size of organs that are so situated as to make palpation impossible.

Technic: The patient assumes a dorsal position with all the muscles relaxed The examiner employs the same technic *for percussing the abdomen* as *is used* in percussion of the thorax, though the stroke is usually lighter and the diagnostic *accuracy necessarily less acute* The note obtained over the normal abdomen is tympanitic, because the greater part of it is occupied by the stomach and intestines, these organs usually containing a sufficient quantity of air or gas to give the abdomen a tympanitic note. The pitch and intensity, as well as the clearness, of this note vary in different regions, depending entirely upon the viscus percussed, its degree of fullness and the admixture of solid material with liquid and air.

Over the small intestines in the umbilical area, the tympany is of high pitch, not quite so loud and clear as it is over the colon. The tympany over an "empty" stomach is much clearer than that elic-

CHAPTER XXI

Examination and Diseases of the Liver, Gallbladder and Spleen

The Liver

Physical Examination of the Liver

The liver is studied chiefly by palpation. Inspection may reveal enlargement in the hepatic region and the condition of the skin, whether it is jaundiced or not; percussion is an aid in confirming and often in elucidating certain signs obtained by palpation, particularly as to size and position; auscultation is of value only in cases where pulsations of the liver are both visible and palpable; auscultatory percussion may occasionally aid in outlining the upper, lower and left borders of the liver, when palpation and percussion yield unreliable information.

The liver is studied as to its (I) contour, (II) position, (III) mobility, (IV) size, consistency, conditions of the surface and edge Associated constitutional symptoms and various laboratory tests are always to be considered when the liver is studied.

I. **Alterations in Contour:** The liver may lose its normal contour because of the presence of some neoplasm upon its surface, such as a cyst, sarcoma, carcinoma, gumma, abscess or other tumor. *Injury to the liver* may change its outline by reason of scar formation. *Pressure* of any kind upon a certain portion of the liver will cause distortion.

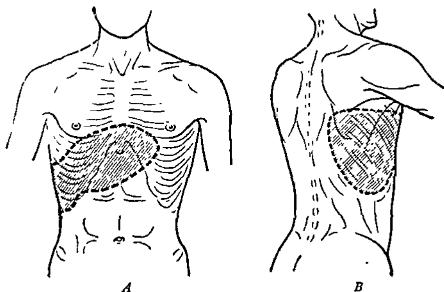


Fig 1—*A*, Surface area of the liver, anteriorly *B*, Surface area of the liver, posteriorly. (After Lejars.)

II. **Position:** The normal position of the liver may be influenced by: (*a*) Conditions in the chest pushing the liver

downward; (*b*) conditions in the abdomen pushing the liver upward, and (*c*) conditions in the abdomen pulling the liver downward.

(*a*) *Conditions in the chest which may push the liver downward* are large pleural effusions, pneumothorax, tumor of

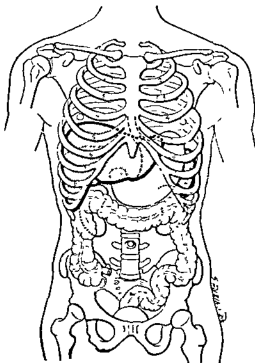


Fig 2—Diagram of the liver, spleen, large intestine, and stomach, viewed anteriorly. (After Letulle.)

the lung, diaphragmatic abscess and aneurysm.

(*b*) *Conditions in the abdomen pushing the liver upward* are large ascites; chronic distention of the hepatic flexure and the colon; acute or chronic peritonitis, and tumors of the kidney; in women pregnancy and ovarian cyst when very large.

(*c*) *Conditions in the abdomen causing the liver to descend* are relaxation of the ligament which holds the liver in position, and general visceroptosis.

III. **Mobility:** A limited amount of motion, *i. e.*, descent during inspiration and ascent during expiration, is normal to this organ. In visceroptosis and long continued ascites a moderate amount of mobility will be found. A true "floating liver" is extremely rare; it may result from a violent injury or a sudden strain such as is induced by vomiting or choking, heavy lifting, or violent coughing; rapid emaciation and tight lacing may also produce a "floating liver"; because of the rarity of this condition it is assumed that a floating liver can occur only when there is congestion tending toward relaxation.

IV. **Size:** Pathologically, the liver may be increased or diminished in size because of disease, though there are a number of diseases to which this organ is subject, in which no appreciable change in its size can be noted.

Diseases of the Liver

Jaundice (Icterus)

Jaundice is classified in three general groups, namely: (I) Obstructive; (II) hemolytic; (III) hepatocellular (toxic, infectious hepatic, suppression jaundice and several subgroups). The degree of jaundice depends upon the amount of bilirubin in the blood. The type of jaundice depends upon the method by which bilirubin has entered the circulation.

Icterus Index: The amount of bilirubin in the blood may be judged by the "icterus index" or the "quantitative Van den Bergh test." The normal icterus index is between 0.1 and 0.5 mg. per cent or about one part of bilirubin in 200,000 parts of blood serum. When the icterus index reaches 1 mg. per cent, a subicteroid tint may be noted in the conjunctivae or skin. Values above 1

mg. per cent become clearly noticeable in the skin, mucous membranes, and the secretions and excretions. The more pronounced the jaundice, the higher is the icterus index (SEE p. 1037).

Van den Bergh Test: The quantitative Van den Bergh test is expressed in units. One unit is equivalent to 5 mg. per 1000 cc. of blood. The normal reading is from 0.2 to 0.6 units per 1000 cc., or 1 to 3 mg. per 1000 cc. of blood. Readings above these values indicate *bile retention*. The icterus index or the quantitative Van den Bergh test when done at regular intervals in cases of jaundice will indicate whether the jaundice is progressive or retrogressive. The method by which the bilirubin has entered the blood is often determined by the qualitative Van den Bergh reaction. The qualitative Van den Bergh reaction *may be (a) direct or prompt, (b) indirect*, (*c*) delayed, and (*d*) biphasic.

The direct or prompt Van den Bergh reaction appears as a bluish-violet color in from 10 to 30 seconds after 1 cc. of serum is mixed with the reagent. This indicates that the bilirubin has passed through the liver cells before its entrance into the circulation and is indicative of *"obstructive jaundice."*

The indirect Van den Bergh reaction is obtainable only when alcohol is added to the mixture of reagent and blood. The color thus obtained, which appears instantaneously, is a violet red. The indirect reaction indicates that the excessive *amount of bilirubin has not passed* through the liver. The liver cells have become incapacitated because of some poison and the bilirubin passes directly into the hepatic veins and thence into the blood stream without first entering the liver cells. This condition is indicative of toxic, or infectious, or hemolytic jaundice.

The biphasic Van den Bergh reaction occurs where both direct and indirect reactions are obtainable; this is found when excessive blood destruction takes place where bilirubin is formed in such large quantities in the reticuloendothelial cells that the liver cells are incapable of utilizing all of it, therefore the excess passes directly into the hepatic veins. It is also indicative of both obstructive and toxic jaundice.

A delayed direct reaction, where the reddish color appears promptly and after standing for some time develops into a violet color, has the same significance as an indirect reaction (SEE: p. 1037).

I. **Obstructive Jaundice:** This type of jaundice results from some *form of* obstruction to the outflow of bile from the liver into the intestines. The bilirubin, which stains all the tissues, passes from the blood through the liver cells into the bile capillaries, but because of an obstruction to the outflow of bile it is *reabsorbed into the blood. Obstructive* jaundice may be caused by: (*a*) Obstruction may occur within the ducts, as in gallstones (cholelithiasis), inflammation of the gallbladder (cholecystitis), echinococcus cysts, and *roundworms* within the gallbladder or gall ducts. (*b*) *Obstruction may* be due to pressure upon the ducts from without, as in malignant tumors of the liver, the head of the pancreas, or the gallbladder, and malignancy of the stomach, causing metastasis to *the glands in the vicinity* of the gallbladder or common bile duct or the hepatic duct. Large tumors of the right kidney, the omentum, the retroperitoneal glands, as well as hepatic abscess, and gumma may cause *compression* of the intrahepatic ducts. In rare cases ob-

structive jaundice may be caused by pressure exerted upon the gallbladder or the liver by fecal accumulation in the hepatic flexure, uterine tumors, and greatly distended pregnant uterus. (*c*) Obstruction may be caused by disease of the walls of the ducts as in cholangitis, choledochitis, injury to the gall ducts, catarrhal jaundice due to swelling of the mouth of the common bile duct, allergic swelling of the bile ducts, infective or suppurative cholangitis, and duodenal catarrh, causing obstruction in the region of the papilla of Vater.

Symptoms: Because of the obstruction to the entrance of bile into the intestines, the bile pigment is reabsorbed from the liver into the blood stream. The skin and mucous membranes become yellow, the sweat and tears are also yellow, but the saliva, cerebrospinal fluid and mucus of the alimentary canal are not bile stained. The urine is very dark because of its bile content When the bile obstruction is complete, urobilin is absent from the urine and the stool is clay colored. The qualitative Van den Bergh reaction is prompt direct. The quantitative Van den Bergh reaction and the color index are high. There is usually itching of the skin; occasionally purpuric spots may appear on the skin and mucous membranes Blood coagulation is delayed and the pulse is slow. The kidney threshold for bilirubin is comparatively low. Bile appears in the urine when the bilirubin concentration in the blood reaches 1 to 50,000

II **Hemolytic Jaundice:** In this type of jaundice the large amount of bilirubin which stains the tissues is caused by excessive destruction of the red blood corpuscles. The hemoglobin thus set free is converted into bilirubin by the reticulo-endothelial system, such as the spleen, the endothelial cells of Kupffer, etc, and not by the glandular cells of the liver Because this type of bilirubin is not a liver product, the qualitative Van den Bergh reaction is indirect. The kidney threshold for this type of jaundice is higher than in the obstructive type Bile may not be detected in the urine until the bile concentration in the blood is very high. The urine is, therefore, not very dark and the stool is very, very dark because of the large amount of bile pigment that finds its way into the intestinal canal by way of the liver, though the liver does not participate in the formation of this type of bilirubin When large amounts of bile pigment occur in the stool and none in the urine, it is known as acholuric jaundice (SEE. p. 559)

The blood destruction occurs chiefly in the spleen, liver, lymph nodes and bone marrow; but with respect to some of the conditions belonging under the head of hemolytic jaundice, we have little knowledge of the place of blood destruction

Two types of hemolytic jaundice are recognized: (1) The acquired type (Hayem-Widal), (2) the congenital or familial (cholemie familiale; Chauffard-Minkowski) In both, far greater amounts than the threshold value of four units of bile pigment may be present in the blood without bile appearing in the urine; hence the synonym, acholuric jaundice. In most cases the bile is excreted in the urine in increased amounts as urobilin and in the feces as stercobilin

The two groups, congenital or familial and acquired, are not often separated since there are many border-line cases, as, for example, congenital cases with negative family history Such cases are

perhaps better classified with the acquired type.

Gallstones are quite common in familial or congenital hemolytic jaundice, but seem to bear no etiologic relation to the jaundice.

One may place under the head of acquired hemolytic jaundice, the icterus found in pernicious anemia and allied conditions in which the Van den Bergh test shows increased value of the icterus index but in which there is no choluria

The cause of hemolytic jaundice is either some defect in the blood, or *some disease of the spleen.*

Icterus Neonatorum: This is a type of hemolytic jaundice due to rapid blood destruction. It may be benign or malignant. The benign form appears in a considerable number of newborn babies during the first few days of life. The grave form of icterus neonatorum is due to sepsis, usually of umbilical origin, to syphilis of the liver, or to congenital absence of the bile ducts. The blood gives a positive indirect but negative direct Van den Bergh reaction

A rare example is the familial type of jaundice of the newborn, a grave disease occurring less often in the children of the first and second pregnancies than in those of later birth. Those that recover often show permanent cerebral or cerebellar defects.

III. Hepatocellular Jaundice (nonobstructive Hepatic Jaundice): Two clinical groups are recognized in this type of jaundice.

1. Catarrhal Jaundice (infectious): This is a type of jaundice occurring chiefly in children and young adults. It may occur in epidemics or singly. It may be due to duodenitis, cholangitis or to acholasia of the bile ducts or of the sphincter of Odi. There may be various degrees of jaundice, enlargement of the liver, and moderate rise in temperature; severe pain is absent.

2. Toxic Hepatic Jaundice (infectious hepatic suppression): This type is caused by certain toxins in the body which destroy the red blood cells and liver cells; and is found in conditions of poisoning by snake venom, chloroform, ether, chloral, potassium chlorate, cinchophen, arsenic and arsphenamine, phosphorus, mercury, arsenobenzol derivatives, trinitrotoluene, tetrachlorethane vapors, sulfanilamide, sulfapyridine, etc It may be caused by overdoses of x-ray or radium

It is also seen in newborn children, pyemia, yellow fever, pneumonia (sometimes), Weil's disease (spirochetosis icterohemorrhagica or leptospirosis), acute yellow atrophy of liver, epidemic influenza, typhoid fever, typhus fever, scarlet fever, relapsing fever and after abdominal operations (rare).

Toxic jaundice may be slight or severe; it is never prolonged because the patient either recovers or dies in a short time. In this disease the feces is not clay colored; in fact, it may be darker than normal, and the urine does not necessarily contain an excessive amount of bile pigment.

Toxic jaundice was formerly classified as hematogenous icterus, while the obstructive variety was known as hepatogenous. This type of jaundice (the hepatocellular) is the commonest variety; it gives a biphasic Van den Bergh reaction because there occurs both blood and liver destruction.

Dissociated Icterus: French writers, and Hoover and Blankenhorn in this country, have called attention to dissociated icterus, that is, one in which the bile salts and bile pigment are separate and do not occur together in the blood

or urine They recognize: (*a*) A hepatic dissociated icterus in which bile salt and bile pigments are separately present in the plasma as the result of separate hepatic excretions into the blood, (*b*) a renal dissociated icterus in which the bile pigment alone is present in the plasma due to renal excretion of the bile salts The subject is one requiring further investigation

Diseases of the Liver Characterized by Enlargement

Normally the liver may be displaced by hydrothorax or pneumothorax and may be mistaken for enlargement. Therefore it is always important to examine the chest when the lower edge of the liver extends beyond the 10th rib anteriorly When the liver is elongated, though otherwise normal, it may extend below the right costal margin

Riedel's lobe of the liver· This is a tonguelike downward projection of the right lobe of the liver which may be mistaken for a displaced or diseased kidney or a tumor. It, however, moves with respiration; is not readily displaced by manipulation; is not tender, and is not associated with enlargement of other parts of the liver.

Enlargement of the liver is observed in· (*a*) Hypertrophic or biliary cirrhosis (Hanot's), (*b*) early stages of atrophic cirrhosis (portal cirrhosis); (*c*) passive congestion (myocardial failure); (*d*) sarcoma; (*e*) carcinoma; (*f*) abscess, (*g*) amyloid degeneration; (*h*) fatty infiltration; (*i*) leukemia; (*j*) echinococcus; (*k*) simple cyst; (*l*) syphilis of the liver; (*m*) actinomycosis; (*n*) tuberculosis of the liver; (*o*) diabetes (rare); (*p*) Weil's disease; (*q*) angioma; (*r*) Banti's disease; (*s*) perihepatitis, early stages; (*t*) hemochromatosis (bronzed diabetes); (*u*) von Gierke's disease; (*v*) Hodgkin's disease; (*w*) acute suppurative cholangitis; (*x*) acute hepatitis; (nonsuppurative); (*y*) obstructive jaundice; (*z*) Gaucher's disease; (*aa*) rickets, and (*ab*) temporarily it may occur in association with febrile and other diseases.

(*a*) **Hypertrophic Biliary or Hanot's Cirrhosis:** *Inspection* will reveal generalized jaundice of the skin, mucous membranes, and sclera; fullness in the hypochondriac region; and dark, bile-stained urine, and clay-colored stool On *palpation*, the edge of the liver will be found hard and rounded, and lying one to three inches below the right costal margin. Its surface will be smooth and resisting, and the left lobe will be palpable as far as the left midclavicular line, and often two to three inches below the lower sternal edge. *Percussion* will often elicit the upper line of dullness as high as the fifth rib, in some instances extending as high as the third intercostal space or fourth rib. Liver dullness at the lower border usually coincides with the palpated lower border of the organ There usually is associated enlargement of the spleen No *auscultatory signs* indicative of this form of liver disease are obtainable.

Symptoms: This disease is insidious in its onset and manifests itself by progressive loss of strength, jaundice, fever at irregular intervals and symptoms of indigestion; ascites is rarely, if ever, present, unless biliary and portal cirrhosis coexist. When it occurs in childhood it is associated with stunted growth, enlargement of the spleen and intense itching.

Pathology: The enlargement of the liver is due to increased connective tissue formation around each single lobule.

hence the name "unilobular cirrhosis." The pathological changes are the result *of contraction of the bile ducts* (for which reason it is often termed "biliary cirrhosis"), and the accompanying jaundice. This may follow chronic obstruction of the bile ducts or chronic infection. It is commoner in males than in females It is a rare disease.

(*b*) **Atrophic cirrhosis of the liver** (portal cirrhosis, Laennec's cirrhosis) is caused by a deposit of connective tissue around the blood vessels, the consequent contraction producing obstruction to the portal circulation During the early stages of atrophic cirrhosis, when the connective tissue is being deposited, the liver necessarily enlarges; but as soon as the connective tissue begins to shrink, the liver is only moderately enlarged, and does not produce any usual symptoms. Pulsations may sometimes be noted When the stage of actual diminution in the size of the liver has taken place, the liver becomes small, *often* bosselated ("hobnailed liver") and presents the following well-known signs, *i e*, ascites, distended veins, caput medusae, hypertension, hemorrhoids and little if any jaundice (SEE. p. 615).

(*c*) **Chronic Congestion or Passive Congestion:** This is due to venous obstruction.

Symptoms: The liver is tender and there is a sensation of fullness and weight *in the hepatic region. In the early stages* there is often expansile pulsation synchronous with the heartbeat. There are signs of venous obstruction, ascites often develops and a mild degree of jaundice and gastrointestinal disturbances are quite common.

Etiology: The commonest cause of venous or passive congestion is back pressure due to heart failure, following regurgitation and failure of the right ventricle. It does not matter which heart valve is the etiological factor in causing decompensation. The heart lesion, most frequently responsible for back pressure sufficient to produce tricuspid insufficiently, is mitral disease. A tumor pressing upon the inferior vena cava above the diaphragm may also bring about passive congestion of the liver.

Diagnosis: On *inspection*, the patient is cyanosed, usually dyspneic and may be slightly jaundiced, the abdomen is enlarged, particularly late in the disease, and the abdominal veins are distended In the early stages the liver is *palpable* a short distance below the right costal border, and is often pulsating In the later stages it is very much enlarged, smooth and presents a rounded edge The liver is tender to pressure, and the lower edge may extend as low as the umbilicus, or even lower, depending upon the severity of the condition and the length of time it has existed In the presence of ascites fluctuation will be demonstrable. It is often difficult to outline the liver by *percussion*, because passive congestion of long standing is usually associated with a right-sided hydrothorax which masks the upper limit of liver dullness, and the lower border is often encroached upon by an accompanying ascites. *Auscultation* is of little value, though *auscultatory percussion* will often give a clue as to the approximate upper and lower borders of the liver.

(*d*) **Sarcoma of the Liver:** This is usually secondary to sarcoma of a bone or *other tissue of the body.* Primary sarcoma of the liver is extremely rare A sarcoma may occur either as a large nodular mass displacing an area of liver tissue or as diffused *infiltrating* growths In the latter type the enlargement is not

as great as it is in the first variety mentioned.

Diagnosis: On *inspection*, the patient, usually a young adult or a child, appears very much emaciated, and often nodules appear on the undersurface of the liver, they are not palpable through the belly wall. Fluctuation is often demonstrable, and the fluid is blood tinged. *Percussion* will aid in

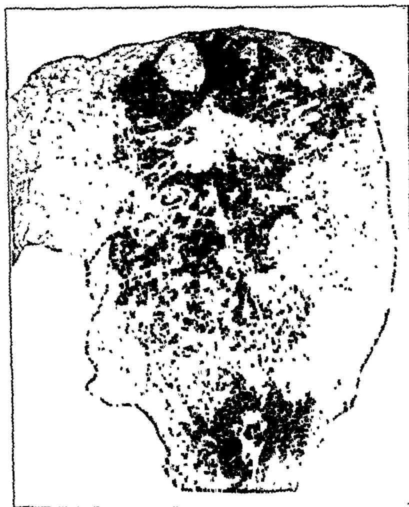


Fig 3—Carcinomatosis of liver

jaundiced and cachectic; in most cases the primary seat of the lesion can be demonstrated. *Palpation* will reveal either a large nodular mass, or numerous small nodules in various parts of the liver which are somewhat tender, but not very painful to touch; when demonstrating the size of the liver. *Auscultation* is entirely negative.

(*e*) **Carcinoma of the Liver:** This is usually secondary to carcinoma of other organs, *e.g.*, the stomach or the gallbladder, pancreas, adrenal, prostate, rectum, uterus, breast, mediastinum,

lungs, kidney, eye, etc. Primary carcinoma of the liver is rare.

Symptoms: In rare cases cancer of the liver may be latent, the patient complaining only of vague pains around the hepatic region, symptoms of indigestion and progressive loss of strength. Usually, however, there is pain or tenderness over the liver, the pain—either dull or sharp—being often referable to the right shoulder.

Diagnosis: On *inspection,* the patient appears thin, emaciated and generally cachectic. There is usually a light yellow tinge to the skin and conjunctivae, and when the bile ducts are affected or there is associated carcinoma of the gallbladder, deep jaundice is the rule. The superficial veins are usually enlarged; puffiness of the lower eyelids and the ankles will be in evidence; this is caused by associated cardiac weakness and anemia *Palpation* will reveal either slight or moderate enlargement, depending entirely upon the position of the carcinoma and the stage of the disease. The surface of the liver may be nodular, the nodes being umbilicated; in cases where there are no nodules, the organ will be hard and unyielding to the touch Ascites is not a common complication, but a small amount of bloody fluid is frequently found There usually is associated enlargement of the spleen. *Percussion* confirms palpation as to the size of the liver and the presence or absence of ascites. Ulnar percussion will elicit sharp pain over the liver region. *Auscultation* is entirely negative

(*f*) **Abscess of the Liver or "Suppurative Hepatitis":** By abscess of the liver is meant an accumulation of pus in the liver tissue. In the majority of cases the condition is the result of some infectious process carried to the liver by the portal circulation. Its etiological factor may be an infectious embolism or thrombus from the lung, suppurative endocarditis, infection by the colon bacilli, or the endamoebae histolytica, and other intestinal parasites. The abscess may be single, multiple, or may occur as a diffuse suppuration

Symptoms: There is sudden, sharp pain radiating towards the shoulder, and often along the diaphragm; this is intensified by pressure, while a change of posture often relieves it. The site of the pain usually depends upon the position of the abscess Chills, fever and sweats are constant symptoms, usually with progressive weakness, emaciation and all the evidences of chronic sepsis. In the presence of amebic abscess diarrhea is a common symptom

Diagnosis: Inspection shows the patient to be anemic and emaciated; jaundice usually develops, particularly when the abscess involves the bile ducts. When the abscess is superficial, bulging over the region where it is located may be noted; if the abscess is subdiaphragmatic, limited right-sided chest expansion will be observed.

On *palpation* the liver is enlarged, and the abdominal muscles over the liver are somewhat rigid; if the abscess is superficial, a soft, somewhat fluctuating mass may be elicited, while if it involves the peritoneal surface, friction fremitus and tenderness may be present; if subdiaphragmatic a tender point may be located in the right upper abdomen

Percussion confirms the palpatory signs of enlargement of the liver. In subdiaphragmatic abscess the descent of the diaphragm as elicited by percussion is much less than on the opposite side.

(*g*) **Amyloid Disease:** Amyloid disease of the liver is usually secondary to

chronic suppuration and is, at times, found in bone tuberculosis and syphilis. It is also found in rickets, carcinoma and is often associated with lymphatic leukemia. In fact, any suppuration, if long continued, may produce amyloid disease of the liver, spleen and kidneys.

On *inspection*, the skin is usually pale and the upper abdomen bulges. *Palpation* shows the liver moderately or enormously enlarged, smooth and firm, with its edge usually rounded and blunt, though in some cases a sharp, well defined margin can be palpated. The liver *is not tender to pressure, nor* does change of posture cause pain. The spleen is proportionately enlarged *Percussion* emphasizes the size of the liver and spleen. Ascites, jaundice and enlarged veins are usually absent.

(*h*) **Fatty Liver:** *Fatty infiltration* consists of an infiltration of fat in the parenchyma of the liver. *Fatty degeneration*, as its name implies, consists of fatty degeneration of the liver structures and usually affects the parenchyma by displacing liver tissue by fat.

Symptoms: These, as a rule, are few and not pathognomonic. The condition is usually found in those inclined to obesity, though it may occur in sufferers from chronic diseases which interfere with oxidation, *e.g*, tuberculosis, certain forms of anemia, malaria, carcinoma, syphilis and phosphorous poisoning

Diagnosis: Inspection usually shows obesity but no alteration in the normal appearance of the skin, no venous enlargements, and no edema. There may be bulging in the liver region due to enlargement. *On palpation*, the liver may be felt as either moderately or enormously enlarged. The surface is smooth and soft and palpation does not elicit pain or tenderness The edge is decidedly thickened and smooth. *Percussion* confirms palpation as to the size of the liver. Ascites is absent. Fatty infiltration is often diagnosed by the presence of a large liver and the absence of other symptoms.

(*i*) **Leukemia:** In myeloid leukemia the liver as well as the spleen is enlarged.

Symptoms: Progressive weakness, pallor, dyspnea, ringing in the ears, and dizziness, often nausea and vomiting, hemoptysis and epistaxis are the most frequent complaints. As the disease *progresses, dimness of vision, severe* anemia, cutaneous hemorrhage, and—in some cases—itching are marked.

Diagnosis: On *inspection*, the skin presents a muddy pallor, accompanied by edema of the face, hands and feet; the abdomen is distended, the greatest distention being noticeable in the splenic region. *Palpation* shows the skin to be rather dry, giving a sense of resistance, and is often edematous The liver may be moderately or enormously enlarged, the usual enlargement, however, being between one and three inches below the costal margin. It is smooth, moderately firm and not painful or tender to the touch. *Percussion* confirms the palpatory signs of enlarged liver

Auscultation is negative, though hemic heart murmurs are frequently heard The spleen is greatly hypertrophied and glandular enlargements in the axillae and groins are common The blood picture is characteristic of the disease (SEE p 566).

(*j*) **Hydatid Cysts:** These result from the lodgment of the *tenia echinococcus larvae.*

Symptoms: General weakness and gastric disturbances are as a rule the only complaints.

Diagnosis: A mass may be visible in the hepatic region, and on *palpation the liver will be found somewhat enlarged.* When the cyst is superficial a soft fluctuating mass can be palpated and, in some cases, several such masses may be found. Aspiration often reveals hooklets in the fluid, which make the diagnosis positive. A diagnosis of hydatid cyst by physical examination alone is impossible, but a history of having been associated with dogs or coming from a locality where the disease is endemic, together with the finding of a soft fluctuating mass upon the liver, and the absence of constitutional symptoms makes the diagnosis of *echinococcus cyst* probable. Very few cases of hydatid disease originating in the United States are on record; most of the patients treated here have acquired the infection in the eastern hemisphere. The *tenia echinococcus* is an intestinal parasite of dogs; it is communicated to cattle and —less frequently—to humans from the dog's excrement, and is most often acquired by humans from eating infected meat or at times direct from the dog. The disease is common in Iceland, Australia, and certain sections of central Europe where dogs live in close contact with humans, and where sanitary conditions are bad. The patient's previous history, therefore, becomes very important in establishing a diagnosis

(*k*) **Simple Cyst:** The symptoms and physical signs of simple cyst are similar to those of hydatid cyst, except that the fluid withdrawn by aspiration does not reveal hooklets or anything that would suggest echinococcus



Fig 4—Polycystic liver.

(*l*) **Syphilis of the Liver:** Syphilis of the liver may occur in those suffering either from the congenital or from the acquired form in the late stages Syphilis of the liver may be of three varieties: (1) Interstitial hepatitis (a diffused inflammatory condition of the liver substance); (2) gumma; (3) perihepatitis

In *interstitial hepatitis* the symptoms are those produced either by pressure or inflammation of the organ On *inspection* the skin is generally jaundiced, and distended veins over the abdomen are quite common. Ascites is not a very frequent complication unless there is interference with the return circulation The liver is usually enlarged, but not to a very great extent.

In the *gummatous* variety, single and, rarely, multiple tumor masses can be *palpated* upon the surface; the most common site being the left lobe and the undersurface of the left extremity of the right lobe, though any portion of the liver may be the seat of a gumma. In the diffuse variety there is usually some tenderness upon pressure. The liver is always enlarged, the left lobe being often disproportionately enlarged, and somewhat irregular in outline, and is firm and tender to touch; signs of general cirrhosis are often found and an associated splenic enlargement is quite common. The diagnosis of syphilis of the liver cannot, however, be definitely established unless a positive Wassermann reaction and other confirmatory luetic evidence can be obtained.

Perihepatitis is an inflammation of the peritoneal covering of the liver, usually occurring in circumscribed areas. It often occurs as an inflammatory extension from a diseased liver and when not due to syphilis it may result from conditions such as abscess and hydatid cyst of the liver, from general peritonitis; or as an extension from pleurisy; or from a perforated ulcer of the stomach, duodenum or gallbladder. Perihepatitis may also be caused by violence, a blow, or any other local injury.

Symptoms: There is usually pain and tenderness over the portions affected Jaundice may occur when the bile ducts are involved, and distended veins and ascites are evident when there is interference with the return circulation

Diagnosis: On *inspection* there may be jaundice, ascites and distended veins, though their absence does not exclude perihepatitis Diminished respiratory mobility will be noted over the right lower chest and upper abdomen. *Palpation* often reveals a friction rub at the junction of the seventh rib and midaxillary line, also, in the midaxillary line at the ninth rib, and occasionally in the epigastrium. The lower edge of the liver is usually palpable, and when pressure is brought to bear upon it, referred pain to the chest will be produced

If suppuration occurs, pus may collect below the diaphragm. On *percussion* chest dullness will be found at a higher level than normal and diaphragmatic descent will be found to be limited.

Before the occurrence of suppuration a friction rub may be *auscultated* over the regions where the "rub" is palpated After suppuration, particularly if it be subdiaphragmatic, all the signs of subdiaphragmatic abscess, such as absence of breath sounds, pain, diminished tactile fremitus, diminished expansion, etc, manifest themselves. An x-ray examination and, at times, an artificial pneumoperitoneum, may assist in arriving at the proper diagnosis.

(m) Actinomycosis: This disease is caused by a ray fungus, *actinomyces* (a streptothrix). When these fungi invade the liver they usually cause multiple abscesses, so that the symptoms and signs of liver abscess are usually found with an associated enlargement of the organ and infection of other parts of the body A positive diagnosis can be made only when the ray fungi are isolated from the aspirated pus

(n) Tuberculosis of the Liver: This is usually secondary to tuberculosis of the lung, bowel, peritoneum, or other structure, or the liver may be one of the organs affected in a generalized miliary tuberculosis, or by a tuberculoma.

Symptoms: There are no symptoms referable to the liver alone. In rare cases

Differential Diagnosis, Disease of the Liver and Its Appendages

Symptoms	Hepatitis	Perihepatitis	Gallbladder (without stones)
Pain, type	Dull aching, constant Referred areas may be present	*More sharp than in* hepatitis. Increased on breathing, on movement and on sitting down with the knees drawn up.	Colic, generally *of paroxysmal* type, suddenly reaching an acme, and then suddenly disappearing, leaving only a feeling of soreness in its place In some cases instead of being paroxysmal the pain may be constant Long intervals of freedom from pain may be present
Relationship to the ingestion of food.	*Worse at the time of* intestinal digestion when the blood content *of the liver is* greatest	*Same as in hepatitis.*	*No special relationship to the* ingestion of food.
Tenderness	Present over liver region.	Present over liver region	Present over margin of gallbladder Murphy's sign present.
Jaundice.	May be present	Absent.	Absent.
Nausea and vomiting	Not specially marked.	Not specially marked.	Generally present May be constant and severe. Bile present
Temperature.	Slight rise	Slight rise.	Septic in cases of inflammation In cases of colic no rise
Pulse	Slight increase	Slight increase.	Considerable increase in cases of inflammation, very slight, if any, increase in cases of colic.
Urine	*Bile may be present*	*No bile*	No bile
Position of election	Pain worse when lying on left side.	On back; breathes very easy	Generally on back, knees drawn up, abdomen relaxed as much as possible
Effect of movement	Increases pain	Increases pain	Increases pain, except in colic
Application of cold or heat	Cold eases pain	Cold eases	Inflammation, cold eases, heat increases Colic, cold increases, heat eases

be differentiated. Fluctuation due to ascites is often present. *Percussion* confirms the palpatory signs. *Auscultation* is *negative* *In the later stages of the disease* there are hemorrhages from the gastrointestinal tract and ascites (SEE: p. 623).

(*s*) **Early Stages of Perihepatitis** (hepatitis externa) Perihepatitis has already been mentioned under syphilis of the liver. Acute syphilitic perihepatitis is, *however, a rare condition. Chronic* hepatic inflammation, with great thickening of Glisson's capsule, is more commonly encountered. Osler and McCrae[1] divide the condition into two groups: One, occurring in adults, presents re-

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when a number of tubercles form near the bile duct and encroach upon its lumen, jaundice may be evident.

Diagnosis: On *inspection*, the patient appears emaciated and has the appearance of one suffering from tuberculosis. The abdomen is usually enlarged and there may be slight jaundice and, at times, also, distended superficial veins *Palpation* reveals that the liver is enlarged, the edge rounded and usually smooth, the surface rather firm, and, in rare cases, very small nodular masses are present. It is neither painful nor tender to the touch. *Percussion* confirms palpation as to the size of the liver If ascites be present, dullness can be elicited in the flanks. *Auscultation* is negative.

(*o*) **Diabetic Liver:** There are cases of diabetes mellitus that do not present an enlarged liver, and an enlarged liver may occur without diabetes. However, in many cases of diabetes mellitus, the liver is found to be hypertrophied so that it may extend to from one to two inches below the right costal margin; the liver is firm, and smooth, the edge is proportionate to its general size; there is no pain or tenderness on pressure and nothing characteristic of the underlying disease may be found in the enlarged liver.

(*p*) **Weil's Disease or Epidemic Catarrhal Jaundice:** This condition is an acute infectious disease characterized by jaundice, high temperature, and enlargement of the liver, spleen and kidney.

Diagnosis: *Inspection* usually shows the patient to be febrile and a moderate degree of jaundice develops on the third or fourth day of the disease. The abdomen is somewhat distended, particularly in its upper half; respiration is shallow. On *palpation* the liver is found to be enlarged, reaching about two inches or more below the right costal border. It is tender to the touch, and at times several tender areas can be definitely outlined. The liver is uniformly hard, and the edge is rounded, smooth and irregular. There is, as a rule, an associated enlargement of the spleen. *Percussion* confirms palpation and may reveal upward extension of liver dullness *Auscultation* is negative, though *auscultatory percussion* may reveal the size of the liver. The *Leptospira icterohemorrhagiae* may be found in the blood and in the urine. Guinea-pig inoculation with the blood may reveal the organism and the characteristic lesions in its viscera.

(*q*) **Angioma of the Liver:** Angioma of the liver is a rare condition, and the diagnosis cannot be made by physical examination alone, though it may be suspected by exclusion. The liver is usually enlarged and, in some instances, the surface is nodular; if the tumor is very large and gives pressure symptoms and every other known condition is absent, angioma may be considered.

(*r*) **Banti's Syndrome** (primary splenomegaly with hepatic cirrhosis, splenic anemia): In this condition the spleen is enormously enlarged, the liver becoming secondarily involved and presenting a cirrhotic condition. It usually affects young people.

Diagnosis: In late cases, on *inspection*, the patient presents the appearance of a grave anemia, the skin is usually jaundiced; ascites is present, and the abdomen is distended. On *palpation* the liver can be felt three or four inches below the right costal margin, and often in such close opposition to the spleen that the inner margins of the two organs can hardly

Differential Diagnosis, Disease of the Liver and Its Appendages

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Relationship to the ingestion of food.	[illegible] greatest	[illegible]	[illegible] relationship to the [illegible] of food
Tenderness	Present over liver region.	Present over liver region.	Present over margin of gall-bladder. Murphy's sign present
Jaundice	May be present	Absent.	Absent.
Nausea and vomiting.	Not specially marked	Not specially marked.	Generally present May be constant and severe. Bile present
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current ascites and symptoms of interstitial nephritis without jaundice, and cannot be differentiated from atrophic cirrhosis of the liver, the other is a manifestation of a widespread fibroid process (multiple serositis) which affects not the liver alone, but may take

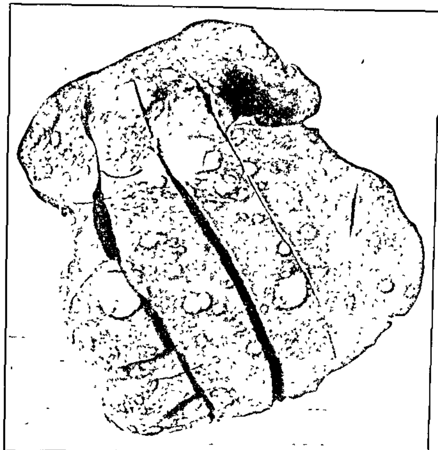


Fig 5—Hodgkin's disease of the liver

the forms of proliferative peritonitis, adherent pericardium, or indurative mediastinitis. Ascites is persistent, and the liver is often smooth and round, resembling the spleen.

(*t*) **Hemochromatosis** (bronzed diabetes): This is a rare disease presenting symptoms of diabetes and hepatic cirrhosis due to the deposit of hemosiderin in the liver and other tissues. There is a bronzing of the skin. The liver and spleen are enlarged and hard. Ascites and enlarged superficial veins are late manifestations.

(*u*) **Von Gierke's Disease** (Hepatomegalia, Glycogenosis): This is a rare disease of childhood characterized by enormous hepatomegaly (due to storage of glycogen), fasting hypoglycemia, infantilism, failure of adrenalin to mobilize glycogen and no splenomegaly

(*v*) **Hodgkin's Disease**: This may at times show enlargement of the liver and spleen or both organs may become infiltrated with tumor masses

(*w*) **Acute Suppurative Cholangitis**: This usually results from obstruction by gallstones, malignancy or parasites; it may also occur in acute infections. It runs

a short course and generally terminates fatally unless interrupted by timely surgical intervention.

Symptoms and Diagnosis: There are jaundice, pain in the hepatic region, signs of sepsis and clay colored stools. The liver progressively enlarges and is extremely tender. The spleen also enlarges. An unfavorable sign described by Rogers consists of the lessening of jaundice and the reappearance of bile in the stool associated with an increase of fever, chills and signs of aggravated infection

(*x*) **Acute Nonsuppurative Hepatitis:** This may be found in catarrhal jaundice and in hepatitis due to arsenic, cinchophen and other drugs and poisons that have a toxic effect upon the liver. There is usually jaundice; very little or no fever; the liver is enlarged, smooth and not especially tender.

(*y*) **Obstructive Jaundice:** Whether due to gallstones, to malignancy of the liver, gallbladder or pancreas, or to other noninflammatory conditions that cause obstruction to the entrance of bile into the duodenum, obstructive jaundice will cause enlargement of the liver, very little tenderness, hardly any fever, but marked jaundice The liver is large, smooth, and its lower edge is rounded.

(*z*) **Gaucher's Disease:** The liver is enlarged but the spleen is very much larger in proportion. There is anemia, a brownish discoloration of the spleen and a characteristically peculiar yellowish wedge-shaped thickening of the conjunctivae on both sides of the cornea The liver is hard, smooth and not tender to touch.

(*aa*) **Rickets:** While normally during childhood the liver is proportionately larger than in adults, and can always be palpated below the right costal angle, in rickets the liver is very large and may occupy the upper right half of the abdomen. It is usually smooth and not tender.

(*ab*) **Temporary Enlargement of the Liver:** The liver may become temporarily enlarged in some of the acute infectious diseases, such as pneumonia, malaria, typhoid fever, scarlet fever, yellow fever, etc The liver usually assumes its normal size when the underlying condition is cured

Diseases Producing Diminution of the Liver

The liver is diminished in size in: (*a*) Atrophic cirrhosis (later stages), (*b*) acute yellow atrophy (acute hepatic necrosis); (*c*) phosphorus poisoning, (*d*) capsular cirrhosis (Glisson's cirrhosis), and (*e*) congenital and chronic acquired syphilis.

(*a*) **Atrophic Cirrhosis or Hobnailed Liver:** In the very early stages the atrophic cirrhotic liver is moderately enlarged, but after the disease has reached an advanced stage the liver begins to shrink, and assumes the characteristic form of this disease.

Symptoms: The initial symptoms are usually vague, but after the disease has progressed for some time, the patient will complain of loss of flesh and strength, morning nausea and vomiting, constipation and hemorrhoids. Often there is epistaxis as well as hemorrhages from the stomach and the bowel. Mental symptoms, clouding of the intellectual faculties, and inability to concentrate are in evidence, and blood pressure is high.

Diagnosis: On *inspection* the veins are usually enlarged, particularly those of the abdomen, and a cluster of enlarged veins around the umbilicus is at times noted (caput medusae). The abdomen is much enlarged and its skin is tense and glistening. The liver cannot be *pal-*

pated at the right costal margin, and ascites is very prominent and easily demonstrable by the presence of fluctuation. *Percussion* usually shows the upper boundary of the liver to be lower than the normal, the lower boundary often being above the last palpable rib.

(*b*) **Acute Yellow Atrophy** (acute necrosis of the liver): This usually results from severe toxemia, such as may be found in pregnancy, infectious jaundice, typhoid fever, influenza, and from the ingestion of certain poisons, such as nonedible mushrooms and other toxic substances. The *liver is small and soft;* it is generally not palpable. The spleen is somewhat enlarged, and it is soft.

Symptoms: These appear in two stages. The first stage lasts from five to six days; in rare cases, it may last several weeks. The onset is gradual with slight fever, jaundice, vomiting, light-colored *stools, and general malaise.* The second stage is ushered in with severe nervous symptoms, such as headache, irritability, delirium, muscular twitchings, and later there may be convulsions *with temporary localized paralysis* resembling meningitis. The vomiting becomes more severe and may contain blood, the stools are clay-colored; the tongue is dry and coated; the pupils are dilated; and the pulse is feeble and rapid. The temperature is subnormal, but may rise just before death. The urine is scanty and contains bile pigment, albumin, casts, and often leucine and tyrosine crystals. The Van den Bergh test gives an immediate direct reaction. Subcutaneous and submucous *hemorrhages occur quite often. Death* may occur within a week after the onset of the second stage.

Diagnosis: The outstanding manifestations are the following: Painless jaundice, mild fever, vomiting, light stools, definite nervous manifestations, small tender liver, petechial hemorrhages, and leucine and tyrosine crystals in the urine.

(*c*) **Phosphorus Poisoning:** This may occur in the employees of match factories or others who come in close contact with phosphorus and inadvertently cause its introduction into the system.

Symptoms: These are epigastric pain, vomiting (the vomitus is black), and nervous disturbances (headache, insom*nia and nausea); delirium sometimes oc*curs in the terminal stages.

Diagnosis: Inspection shows jaundiced skin and mucous membranes. *Palpation* in acute phosphorous poisoning reveals the liver to be enlarged, while in chronic phosphorus poisoning the liver is small, and can be palpated only during *deep inspiration; it is tender to the touch,* but handling it does not cause severe pain. *Percussion* confirms the palpatory signs as to the size of the liver. Care should be taken to differentiate between chronic *phosphorus poisoning and acute yellow* atrophy of the liver.

Differential Diagnosis: This depends upon the history, the type of vomitus and stool, and the presence of petechial hemorrhages.

(*d*) **Cruveilhier-Baumgarten Syndrome:**[1] This is characterized by portal hypertension with excessive umbilical circulation, patency of the umbilical vein, atrophy of the liver with little or no cirrhosis, and splenomegaly. It is believed to be due to congenital patency *of the umbilical vein,* together with hypoplasia of the portal system.

[1] Armstrong, E. L., Adams, W. L., Tragerman, L. J., and Townsend, E. W.: Ann. Int. Med., 16:113 (Jan.) 1942.

(*d*) **Capsular Cirrhosis:** This term is applied to a form of perihepatitis in which the capsule is very hard, thick, and almost semicartilagenous in appearance, the capsular hardening causing a shrinkage and irregular distortion of the liver. The liver, if at all palpable, is smaller than normal, and the edge is irregular, hard and serrated.

Symptoms: Symptoms and physical signs of capsular or Glisson's cirrhosis are very much like those of atrophic cirrhosis of the liver.

Diagnosis: This is based on a positive Wassermann reaction, a small irregular liver and pain in the right upper quadrant. Jaundice and ascites may coexist early in this condition; often, however, it is diagnosed only on the post-mortem table.

(*e*) **Congenital Syphilis and Chronic Acquired Syphilis:** These usually cause a small liver, as the result of shrinking of the deposits of connective tissue within the liver substance. The symptoms and the physical signs are similar to those of atrophic cirrhosis of the liver.

The Gallbladder

Physical Examination of the Gallbladder

The normal gallbladder because of its structure and anatomical position does not lend itself to physical examination.

By cholecystography the gallbladder may be outlined and a general idea obtained as to its function and often the presence of calculi may be discovered. The bile may be obtained by duodenal drainage and examined by chemical and microscopic means

The pathologic gallbladder, when inflamed or enlarged, may be detected by physical examination. An inflamed gallbladder may be suspected by the elicitation of tenderness in the gallbladder region both by palpation and by ulnar percussion. An enlarged gallbladder may be palpated as a rounded, often tender and, at times, fluctuating mass beneath the lower edge of the liver on a line corresponding to an extension of the right midclavicular line. The mass usually moves downwards with inspiration and upwards during expiration. As a general rule the upper portion of the right rectus abdominis muscle is rigid. For proper gallbladder palpation, the patient is to assume the recumbent posture, shoulders raised and knees somewhat flexed. The examiner should palpate lightly with his finger tips so as to elicit resistance, then more deeply in an attempt to outline the shape of the gallbladder, its consistency and the presence of tenderness. The pathologic gallbladder should also be studied by x-rays (cholecystography) and an attempt should be made to study the bile (See: p. 986).

Diseases of the Gallbladder

Cholecystitis: Inflammation of the gallbladder may be due to the presence of gallstones, bacteria, parasites or organic and inorganic material. The infection may be blood-borne and, in that event, first affects the walls of the gallbladder, causing an interstitial cholecystitis. Inflammation of the gallbladder may also be caused by extension of inflammation or growths from adjacent organs, *i.e.*, from the duodenum, pancreas, gall ducts, stomach, liver, etc. The bile is usually concentrated and some-

Differential Diagnosis, Gallbladder Colic a

Symptoms	Gallbladder Colic	
Pain.	[illegible]	L
Jaundice	Generally absent. This is especially true should the cause of the gallbladder colic be an obstruction in the cystic duct.	G
Local tenderness	Higher in the epigastrium and more toward the costal arch than is the tenderness associated with gall-duct colic.	At
Vomiting	Common and continued after the first paroxysm Generally no bile.	G b
Tumor.	Always present, is movable if adhesions are not present	No

Gallbladder and gall-duct colic are often so intimately associated ... distinguish between the two The gallbladder colic is almost an i... sequel of gall-duct colic

times the gallbladder may become distended and give rise to pain and to tenderness on palpation. When obstruction occurs jaundice is a common symptom.

Acute Cholecystitis: This is characterized by pain, tenderness and rigidity in the gallbladder region Pain is often referred towards the right shoulder, to the spine or to the right interscapular region Nausea, vomiting, irregular fever and occasionally jaundice are present; in a thin subject a mass may be palpable in the gallbladder region.

Cholelithiasis: Gallstones may remain dormant in the gallbladder for some time and give rise to very few symptoms such as slight digestive disturbances and a sense of heaviness in the right hypochondrium, or gallstones may cause a great deal of distress by bringing about inflammation and distention of the gallbladder which will give

rise to tendern... severe gastric d... out jaundice. ... pass through t... struction they ... colic which are ... agonizing pain ... drium or epiga... back and right ... usually comes ... meals, as a rul... empty, which ac... most of the atta... cur during the n... to the outflow o... jaundice manifest... derness in the gal... sociated with vo... acidity. The gallb... distention, may be...

Cholangitis: ... gall ducts may be ... tive.

ease, though leukopenia is the rule. The skin is somewhat jaundiced or brownish, most noticeable in the exposed surfaces, but the mucous membranes are not affected. Usually a brownish pinguecula is noted on the nasal sides of the conjunctivae. Gaucher's disease is often accompanied by congenital malformations such as multiple cysts of the spleen and ovaries, horseshoe kidney and patulous foramen ovale. The ante-mortem diagnosis of this condition is based upon the enlargement of the spleen and liver, mild discoloration of the skin, absence of anemia, presence of leukopenia, pains in the muscles of the legs and by the results of splenic puncture

(*h*) **Amyloid Disease:** This causes enlargement of the spleen, liver and kidneys It is usually associated with long *standing suppuration, malignancy or tuberculosis.* The spleen becomes very large, is smooth and uniformly resistant The enlargement of the spleen is only an incident in the disease and alone bears no diagnostic feature but size and smoothness, which are conditions prevalent in other types of splenomegaly.

(*i*) **Pernicious Anemia:** In this blood disease the splenic enlargement is part of the symptom complex The spleen is usually enlarged to about one or two inches below the left costal margin; it is smooth and painless to the touch During the remissions of the anemia, the spleen diminishes in size only to reënlarge during an exacerbation



Fig 7—Adenocarcinoma of spleen.

(*j*) **Cysts:** This is a rare condition; it may be single or multiple. The commoner cysts of the spleen are echinococcus (hydatid) dermoids or cystic degeneration. The spleen becomes enlarged often in proportion to the size of the cyst. When the cyst is superficial and the abdomen is not unduly distended and the abdominal wall is not rigid or fat, the cyst may be palpated as an *elevated mass upon the surface of the* spleen and when the cyst is very large and not too tense, fluctuation may be elicited

(*k*) **Syphilis:** This may involve the spleen alone, but usually the spleen and liver are *simultaneously affected* The spleen becomes large. Ascites, jaundice,

be a combination of structures leading to a diagnosis of sarcocarcinoma

(*f*) **Splenic Anemia** (not of the Banti's type): By this term is meant a disease of the spleen resulting in a general anemia. It is doubtful if such a disease entity really occurs. There are numerous blood diseases and anemias that are associated with an enlarged spleen as for instance, myelocytic leukemia, Banti's disease, pernicious anemia, malignancy, Hodgkin's disease, etc., in which the splenic enlargement is only one of the symptoms. However, there are cases of severe secondary anemia that present no other definite blood findings than those found in grave secondary anemia and no other physical signs except an enlarged spleen, all other findings being negative. Such cases for the want of a better name are styled splenic anemia.

(*g*) **Gaucher's Disease**: This is usually a familial disease that manifests itself chiefly in the female at the time of puberty or earlier. The spleen becomes enormously hypertrophied. Histologically it is characterized by the presence of Gaucher cells in the reticulo-endothelial system of the spleen and often in other tissues. In the infantile variety of this disease, the bone marrow and often the skeleton may be infiltrated with Gaucher cells. The liver is also enlarged and contains Kupffer cells. Anemia is manifested fairly late in the dis-

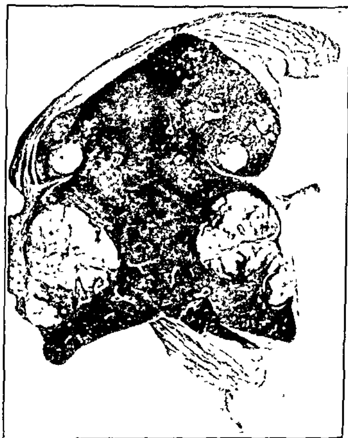


Fig. 6—Sarcoma of spleen

(*p*) **Kala-azar:** This is a tropical disease and is characterized by secondary anemia and marked enlargement of the spleen which harbors the Leishman-Donovan bodies (SEE: p. 1069).

(*q*) **Bronze Diabetes** (hematochromatosis): This is often associated with Banti's disease. It is a condition in which hemosiderin is deposited in the tissues, causing a brownish discoloration of the skin The spleen is large and hard to the touch, the liver and pancreas are cirrhotic, and the urine and blood contain an excess of glucose.

(*r*) **Enlargement Without Any Apparent Cause:** This is often noted. Such cases may be due to chronic infection or to illness of long duration, the spleen having failed to resume its normal size after the underlying disease has been cured.

(*s*) **Irregular Enlargement:** This may occur as a result of carcinoma or hydatid cyst.

(*t*) **Gradual Hypertrophy:** Of varying degrees, this occurs in amyloid disease, pernicious anemia, congestion due to portal obstruction, rickets, splenic capsulitis and splenic infarcts.

(*u*) **Enlargement of Both Liver and Spleen:** This may occur as a result of passive congestion, cirrhosis, hydatid infection, leukemia and amyloid disease, pseudoleukemia (Hodgkin's disease), malarial cachexia, Gaucher's and Niemann's splenomegaly. The associated symptoms and the laboratory findings will help in the differentiation of these conditions. The cause of splenic enlargement cannot, as a rule, be determined by the physical examination of that gland alone It usually requires a complete physical examination of the patient supplemented by certain laboratory examinations.



Fig 9—Hodgkin's disease of the spleen.

(*v*) **Rickets:** This may be diagnosed by its characteristic deformities. The spleen is hard and may be palpable for two fingers' breadth below the costal margin.

(*w*) **von Jaksch's Anemia** (pseudoleukemica infantum): The spleen is hard and may reach the umbilicus.

(*x*) **Hodgkin's Disease:** The spleen may be palpable one or two fingers' breadth below the costal margin.

(*y*) **Congenital Family Cholemia** (acholuric family jaundice): The spleen

frequent hemorrhages in the skin, hemoptysis, hematemesis and melena may occur and secondary anemia is the rule. Syphilis of the spleen may be suspected when the aforementioned symptoms occur in the presence of a positive Wassermann and other manifestations of syphilis It should be borne in mind that a patient may have a splenomegaly and a positive Wassermann reaction, both being due to different etiologic factors.

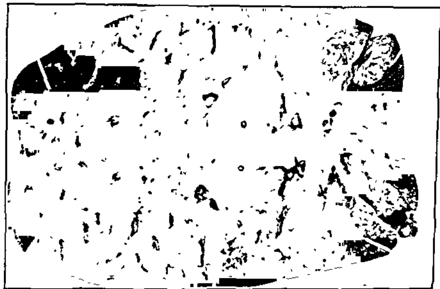


Fig. 8—Tuberculosis of spleen

(*l*) **Leukemia:** The myelogenous or splenomedullary type of leukemia has as one of its characteristic physical findings an enormously enlarged spleen which is hard and of uniform density. The blood findings are usually sufficiently pathognomonic to decide the diagnosis. In lymphatic leukemia also, the spleen is at times enlarged to some extent, though it never becomes large enough to constitute a major sign. *A combination of* myelogenous and lymphatic leukemia has been described in which the spleen attains *quite a large size*

(*m*) **Tuberculosis:** This usually occurs in association with tuberculous peritonitis, glandular tuberculosis, acute miliary tuberculosis and seldom as a primary infection. The spleen becomes *progressively enlarged, is tender to* palpation and often becomes irregular in outline. It is associated with a septic *temperature, cyanosis, polycythemia and* a positive von Pirquet.

(*n*) **Niemann-Pick's Disease:** This is a *condition closely resembling* Gaucher's disease. The spleen and liver become enormously enlarged, the skin usually presents a brownish discoloration, the tongue is geographically coated and the person so affected, usually a child, develops Mongolian features. The blood count shows no anemia but, as a rule, a leukocytosis, the lymphocytes often preponderating and the blood platelets being greatly diminished in number, frequently as low as 20,000 per cubic millimeter. The blood contains an excess of *lipoids.* The large spleen on section presents small white areas which contain special *reticulated cells (foam cells)* that possess phagocytic action. This disease is also known as *lipoid histiocytosis*

(*o*) **Splenomegaly with Eosinophilia:** This condition is rare; the spleen is markedly enlarged and the blood presents a leukocytosis of 34,000 with 70 to 80 per cent of eosinophils and embryonic red corpuscles.

CHAPTER XXII

Examination and Diseases of the Esophagus, Stomach and Pancreas

The Esophagus

Physical Examination of the Esophagus

The esophagus does not lend itself to examination unless special technic has been acquired by the examiner A stricture of the esophagus may be explored by the esophageal sound, a dangerous instrument in the hands of the untrained Esophagoscopy may reveal the appearance of the mucosa and detect ulcerations, varicosities, and growths; a radiogram may show constriction and dilatations Fluoroscopically, a stricture of the esophagus may be recognized by watching the course of an opaque substance during the act of swallowing.

Diseases of the Esophagus

1. *Spasm of the Esophagus (Esophagismus)*

This is a functional constriction of the esophagus causing difficulty in swallowing It is occasionally associated with severe retrosternal pain, referred pain in the pectoral muscles is more common

Cardiospasm associated with spasm of the lower end of the esophagus may cause a large saccular or fusiform dilatation of the lower end of that tube

Diagnosis: The patient is usually a neurotic who may present spastic symptoms in other organs A definite diagnosis may be made when the patient is asked to swallow a capsule containing an opaque material and the passage of the capsule into the stomach is observed under the fluoroscope An esophageal bougie or a large stomach tube may be passed down the esophagus for diagnostic purposes. If the encountered stricture disappears after taking large doses of belladonna or any other antispasmodic, a diagnosis of esophagismus may be made.

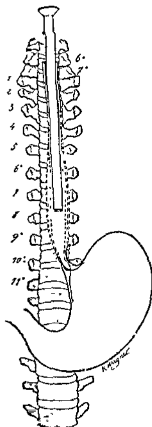


Fig. 1—In the living subject, the lower three-fourths of the esophagus constitute, not simply a canal, but an actual expanded cavity.

is considerably enlarged, and there is an absence of bile in the urine though the stool contains much bile. There is also secondary anemia and fragility of the erythrocytes.

(*z*) **Status Thymicolymphaticus:** There is a general lymphatic hyperplasia. The spleen is moderately enlarged, but is not tender.

(*aa*) **Reticuloendotheliosis:** The reticuloendothelial system is made up of a special kind of epithelial cells often termed *fixed wandering cells* which, though widely distributed throughout the body, possess in common highly specialized functions. These cells are differentiated from other epithelial cells by their ability of being stained *in vivo*, that is, when a dye such as carmin, trypan blue, etc., is injected into the circulation of a living animal, these cells take up the dyes by phagocytic action and are deeply stained by them.

Distribution: They are found in their greatest number in the spleen; they are also found in the liver (Kupffer cells), the bone marrow, lymph glands, the lining of blood vessels and, to some extent, in the adrenal and pituitary glands

The cells comprising the reticuloendothelial system are: (1) The reticuloendothelial cells proper may either be attached to the reticulum of the hemopoietic organs, or may line the sinusoids of certain organs (2) The histiocytes, the phagocytic resting wandering cells, constitute the essential part of the spleen; they are the most active phagocytes in the body, disposing of bacteria and other foreign substances They are also greatly concerned with the metabolism of fat, lipoids, hemoglobin and iron which are stored within them The cells containing an excessive amount of lipoid have a foamy appearance and are known as "foam cells." (3) The monocytes are large mononuclear leukocytes derived from the endothelial tissue of the reticuloendothelial system and have great phagocytic powers. (4) The microglia of the brain and spinal cord, because they develop into large phagocytic cells, are considered by Boyd to belong to the reticuloendothelial system.

Function: The functions of the cells comprising the reticuloendothelial system are phagocytosis, storage of fat, antibody formation, destruction of erythrocytes and the formation of bilirubin.

Diseases of the Reticuloendothelial System: A number of diseases, some of them familial, which present a large spleen, the presence of foam cells containing *kerasin* in the spleen, liver and bone marrow and which show other evidence of disturbed lipoid metabolism are ascribed to disturbed function of the reticuloendothelial system These are Gaucher's disease (SEE p 624), Niemann-Pick's disease or lipoid histiocytosis (p. 626), Hand-Schuller-Christian's disease (p 771), xantomatosis (p 771), and monocytic leukemia or leukemic reticulosis (p 568).

Other diseases suspected of being due to dysfunction of the reticuloendothelial system are infectious mononucleosis (p 569) and Hodgkin's disease (p 569).

Reticuloendotheliosis of the Spleen: This term is applied to a number of ill-defined conditions of unknown etiology running a chronic course in which there is enormous enlargement of the spleen often associated with an enlarged liver This is associated with a severe anemia, leukopenia, and occasionally with monocytic leukemia and with extreme hyperplasia of the reticuloendothelial tissue of the spleen, liver and bone marrow which contain numerous foam cells. A similar condition may at times be produced after prolonged radium treatment

Reticulum Cell Sarcoma: This may occur as a primary neoplasm in the spleen, the lymph nodes, the bone marrow or in other sites where the reticuloendothelial cells are found in abundance The tumor is highly malignant.

Diagnosis: The diagnosis is based upon the age of the patient, dysphagia, emaciation, and esophagoscopic and x-ray findings

5. *Rupture of the Esophagus*

Etiology: Esophagomalacia (softening of the esophageal wall), weakening of the wall near an ulcer or a cicatrix;



Fig 3—X-ray picture of carcinoma of the esophagus showing stenosis (Courtesy of Leon Solis-Cohen.)

also when a great strain is brought to bear upon the weakened wall, by violent and persistent vomiting after a large meal, during acute alcoholism, or in an opium addict because of the sudden withdrawal of opium.

Symptomatology and Diagnosis: The characteristic symptoms are nausea, severe vomiting of the stomach contents and blood, sudden sharp retrosternal pain, pneumothorax and collapse, which may at times simulate angina pectoris or gastric ulcer. Subcutaneous emphysema of the neck and chest may occur in rupture of the esophagus, and is absent in angina pectoris and gastric ulcer.

6. *Dilatations and Diverticula*

These may occur singly or multiply as circumscribed pouchy dilatations of the esophageal wall, and may be situated in the upper portion of the tube or near its entrance into the stomach, the latter as a result of cardiospasm.

Etiology: These may be congenital or acquired: When *acquired* they may be caused by (*a*) pressure from within, and are found on the posterior wall, and (*b*) by traction from without by constricting adhesions; these, as a rule, are found on the anterior wall.

Symptomatology: The symptoms usually consist of a sense of fullness in the sternal region, the sensation of a "lump in the throat" after meals, regurgitation of small quantities of food after strenuous muscular work, particularly on heavy lifting or bending over, and a fetid odor on the breath. When the diverticulum is large, vomiting of large quantities of undigested food that was taken possibly several days previous is noted. Pressure symptoms and changing physical signs from dullness when the diverticulum is filled with food or fluids to resonance when it is empty is a valuable sign Soon after vomiting, tympany may be elicited over a large diverticulum A correct diagnosis of this condition may be made only by x-rays and fluoroscopic studies

7. *Plummer-Vinson's Syndrome*

This is a type of secondary anemia associated with dysphagia, particularly for solid food, and glossitis (See: p. 556).

2. *Acute Esophagitis*

This is an acute inflammation of the esophageal mucosa or of its entire structure.

Etiology and Symptoms: (*a*) Swallowing of irritating substances (lye, acids, mercury, arsenic, hard foreign bodies, *i. e.*, glass, nails, stomach tube, and hot foods).

(*b*) Extension of inflammation from pharynx, larynx, bronchi and mediastinal tissue.

(*c*) Acute and septic fevers (typhoid, typhus, smallpox, diphtheria)

(*d*) Local disease — carcinoma of esophagus or adjacent tissue, vertebral or glandular abscess and laryngeal perichondritis.

(*e*) Spontaneously in sucklings.

The symptoms are pain on swallowing, particularly of hot drinks or diluted alcohol, tenderness over sternum and at times vomiting of blood, pus, or both.

3. *Stricture of the Esophagus (Stenosis of Esophagus)*

Etiology: (*a*) Acute esophagitis; (*b*) cicatrix of a healed ulcer (after lye, bichloride of mercury or other corrosives); (*c*) gumma or its resultant cicatrix; (*d*) congenital stenosis; (*e*) constriction from within the lumen—carcinoma of the esophageal wall, abscess or papilloma; foreign bodies partially obstructing the lumen; (*f*) compression from without by tumor, abscess, aneurysm, enlarged lymph glands, enlarged thyroid, angioneurotic edema (transient), huge pericardial effusions.

Symptoms: The symptoms are gradually increasing dysphagia, regurgitation of food, either immediately after eating when the stricture is high, or some time after swallowing when the stricture is low, accompanied by esophageal dilatation above the site of stricture. Rapid loss of weight may occur as a result of the inability of food to reach the stomach.

Diagnosis: The diagnosis as to the site of the lesion can be made only by esophagoscopy and x-ray examinations



Fig. 2—Carcinoma of esophagus.

4. *Carcinoma of the Esophagus*

This disease may affect any portion of the tube and is a frequent cause of esophageal obstruction in old people. It often causes ulcerations and metastasis to the trachea, larynx, lungs and other structures.

Symptomatology: Swallowing becomes increasingly difficult and is often associated with pain and a choking sensation; there is regurgitation of food and drink; progressive emaciation takes place as the stenosis becomes more marked, and is associated with general cachexia and anemia; at times, notwithstanding the anemia, the blood cell count may be high because of dehydration.

hernia or an acute obstruction. If the mass is pulsating, it may be due to aneurysm of the aorta, or of the celiac axis. A deep-seated tumor in this region may be a growth on the pancreas (For swelling or tumors of the abdomen regionally described, see p 591.)

Percussion: This is employed in order to ascertain the shape and the position of the stomach Care must be taken to note the degree of distention of the bowel and stomach, because very often percussion of a distended transverse colon and an empty stomach may give erroneous results Again, a stomach that is half filled with food, or one that is entirely filled, will give erroneous estimates as to its size.

Auscultatory percussion will, in the hands of experienced observers, give more accurate data as to the size of the stomach than will ordinary percussion

When the stomach is *auscultated*, various crackling, rumbling or gurgling sounds and succussion splashes can be heard, but their significance as to disease of the stomach is of doubtful value (For the significance of the stomach contents, see p 1028.)

Symptomatology of Stomach Diseases *(See: p. 90)*

In a discussion of diseases of the stomach, even in so brief a chapter as this, it is necessary to call attention to the many "gastric symptoms" that may be of extragastric origin Thus we find that diseases of the liver, gallbladder, appendix, bowel, pancreas, heart, lungs (tuberculosis), brain, sinuses, eyes, nose and throat, thyroid, kidneys, the blood and also various constitutional and nervous diseases, such as anemia, fevers, septicemia, helminthiasis, chronic intoxication, diabetes, tabes dorsali, sclerosis of the abdominal vessels, neurasthenia, hysteria and often pregnancy will cause patients to complain chiefly of "indigestion." It must be remembered, however, that a nervous patient, or one who is suffering from one or more of the conditions mentioned, may also be suffering from an organic disease of the stomach, such as ulcer or cancer, and the nervousness, anemia or other conditions may be the result of ulcer or cancer. Therefore, when a patient complains of "gastric symptoms" which may appear to be of extragastric origin, he should nevertheless receive a very careful and thorough gastric study.

When eliciting a history of a patient indicating digestive disturbances, it is well to bear in mind the series of questions tabulated by J M. Anders.[1]

Pain: When pain is present, it may be located at the pit of the stomach (*cardialgia*), or in the gastric region (*gastralgia*). The pain may be severe, slight, or merely a discomfort and uneasiness. All important is it to know when and how (sudden or gradual) the pain appears, and what conditions excite or relieve such distress Does the pain develop before mealtime and when the stomach is empty, and is it appeased by the taking of food; or is it excited by taking food, and does it appear immediately after food, or one to four hours later? Is the pain constant, and is it local or diffused? Does it radiate to the back or scapular regions?

Appetite: The loss of appetite (*anorexia*) or a desire for unusual foods, *parorexia*, are frequently noted. When the appetite is increased, or the patient becomes hungry a short time after a meal, it is referred to as "*bulimia.*" One

[1] Anders, James M.: Practice of Medicine, 14th Edit, W. B. Saunders Co

The Stomach

Physical Examination of the Stomach

Diseases of the stomach are investigated by physical signs, laboratory examination of its contents and by the x-rays. The principal object of the physical examination of the stomach is to determine its size, position, the presence or absence of a tumor mass, tenderness and pain upon pressure.

Inspection: The size and the position of the stomach can only be determined when it is greatly distended with gas. A stomach so greatly distended with gas that it is recognizable by inspection of the abdominal wall is usually in an abnormal position and in a state of great tension. Inspection is only of minor value in determining the degree or absence of peristalsis; a large mass in the epigastrium, however, calls for a thorough investigation by other physical means. A distinct bulging in any part of the abdomen except in the epigastric region may be due to a dilated stomach, such bulging being most frequently noted in the hypogastric or umbilical regions, the epigastrium exhibits a hollow or a transverse depression. A marked depression between the costal arches in the lumbar region, accompanied by a vertical median sulcus, wider above than below, and the abdomen being flattened in the central portion and bulging in the lateral region, is significant of gastroptosis

Palpation: This is employed to elicit tenderness, resistance, tumors, and succussion splash.

The presence of *tenderness* in the epigastric region may denote gastric ulcer, gastric carcinoma, or acute or chronic inflammation of the stomach. The tenderness produced by a gastric ulcer is localized at a definite point and is persistent. A tender point near the left tenth or eleventh dorsal spine is often significant of gastric ulcer.

Resistance over the stomach may be caused either by rigidity of the recti muscles, or the existence of some underlying solid mass. Resistance in the epigastrium may be caused by the enlarged left lobe of the liver, local peritonitis due to perforated ulcer, inflammation or tumors of the omentum, and carcinoma of the stomach, at times also a growth on the pancreas may be mistaken for a gastric condition. Resistance in the umbilical region may be due to a dilated and distended stomach, peritonitis, tuberculosis or cancer of the omentum, or to a displaced organ, such as the spleen, liver, or a greatly enlarged movable kidney.

Pelvic tumors, such as a pregnant uterus and ovarian cyst, may reach the liver and overlie the stomach, thereby making stomach palpation impossible. The normal stomach can be palpated only when greatly distended with gas or air. The old, rather dangerous method of inflating the stomach with a seidlitz powder will bring out its contour so that it can be easily palpated

Tumors. Benign tumors of the stomach are extremely rare A tumor palpated in the epigastrium in an elderly person usually means carcinoma; in young persons, a tumor in the epigastrium or a little below, may be caused by hypertrophy of the pylorus, or by adhesions due to some inflammatory condition. A soft, nonresisting tumor mass may result from dilatation of the stomach or of a portion of the bowel, an omental

spasm of the pylorus and cardia) is often seen in air swallowers.

Belching may be caused by gastric fermentation, swallowing of gas-containing food or drinks, imbibing simultaneously of acid and alkaline food or drink, and air swallowing. The "gas" brought up by air swallowers is odorless and tasteless

(*b*) ***Gastric Carcinoma:*** Vomiting may occur at varying intervals after taking food; it is believed that the closer the lesion is to the cardia the sooner will vomiting occur after feeding. When the lesion is at the pylorus, vomiting may be delayed several hours In carcinoma attended with gastrectasis, vomiting may occur six to twelve hours after tak-

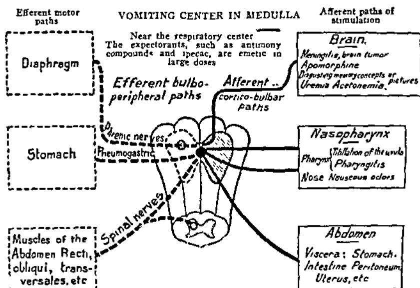


Fig. 4—Pathogenesis of vomiting.

Emesis

(SEE. p. 91)

Vomiting may be of: (I) Gastric; (II) systemic; (III) nervous, and (IV) reflex origin; (V) it may also be caused by direct irritation of the vomiting center

I **Gastric Origin** (organic lesion in the stomach): (*a*) ***Gastric Ulcer:*** Pain and vomiting occur soon after the taking of food, when the ulcer is at the fundus; one or two hours after the taking of food when the ulcer is at the pylorus. The pain stops after vomiting. The vomitus is sour smelling and often contains blood.

ing food. At times vomiting will occur when the stomach is empty In some forms of carcinoma (carcinoma of fundus or lesser curvature) vomiting may be absent. The vomitus is usually sour and has a characteristic odor. Coffee ground vomit occurs when the carcinomatous tissue ulcerates and causes bleeding.

(*c*) ***Acute Gastritis:*** Vomiting of gastric contents, mucus and bile is followed by a sense of relief.

(*d*) ***Chronic Gastritis:*** Vomiting occurs at various intervals after the taking of food. The contents are partially digested food, large quantities of mucus and often bile.

should determine further whether the appetite comes on when the patient begins to eat, or disappears at the sight of food, or after a few mouthfuls of food are taken. The taking of abnormally large amounts of food at mealtimes only is termed *"polyphagia"* Where the appetite is not satiated, even after a full meal, we refer to such condition as *"acoria."*

Thirst: In certain maladies, the thirst is increased, while in a second class of conditions there is little or no desire for water or other liquids. Inquire whether thirst is allayed by taking water.

Taste: Many gastrointestinal conditions are accompanied with an unpleasant, sour, bitter or sticky taste which may be experienced only on waking, or it may be more or less persistent.

Deglutition: Does the patient swallow both solids and liquids naturally, also is he liable to cough while eating, and does such effort cause discomfort or pain?

Pyrosis: This is a burning sensation in the epigastrium and sternal region. Note at what time, before or after food, it is experienced, its duration and how is it influenced by various foods.

Regurgitation: Note how long after taking food this annoying symptom is observed, and also whether the food tastes sour. When the contents of the stomach are expectorated, it is referred to as *regurgitation,* but should it be again chewed and swallowed, it is termed *"rumination"*

Hiccup: The time at which hiccup occurs, and whether or not it is accompanied with a burning sensation in the throat or by an unpleasant odor, are points of clinical value. Prolonged hiccup is of grave significance.

Nausea: *Is it occasional or frequent,* and how influenced by food and by sleep?

Vomit: Inquire carefully as to the frequency of the vomiting; how influenced by pain; when the stomach is empty, after soft food, solid food, or is it excited by certain odors? The quantity and consistency of the vomit, as well as whether it ever contains fresh blood (red), or blood that has lingered in the stomach for a time (coffee-brown vomit)?

Such special symptoms as constipation, mental dullness, sleepy and giddy sensations and a blurring of objects are not infrequently observed in gastric disorders.

Appetite

Appetite may be defined as the relish for food, though not necessarily hunger (SEE · p. 89)

Disturbance of Motor Function

The stomach usually empties itself in from three to six hours, depending upon the kind and quantity of food ingested

Hypermotility (increased peristalsis) may occur in nervous individuals, in hyperchlorhydria, gastric irritation by indigestible food, achylia gastrica, diarrhea, nonobstructive gastric ulcer, gastric carcinoma with achylia and gaping pylorus (Stevens) and in congenital pyloric obstruction.

Hypomotility (diminished peristalsis with atony) is found in gastrectasis, pyloric obstruction due to carcinoma of stomach, stricture, adhesions and persistent pylorospasm.

Pneumatosis (hyperdistension of the stomach with air, due to simultaneous

tumors, and (*c*) by *chloroform*, or *ether narcosis* may cause vomiting.

Characteristics of the Vomitus: When examining the vomitus it is important to note its general appearance, consistency, color, contents, quantity, odor and reaction.

The ***general appearance*** depends upon: The kind of food or other material swallowed; the lapse of time between food taken and its being vomited; and the presence in the stomach of mucus, blood, coloring matter, saliva, acids and foreign bodies.

When vomiting occurs soon after eating, the food will show very little change; after the lapse of an hour or two, the food will show partial digestion; five or six hours later, no food should be found in the vomitus In retention vomiting, i. e., gastrectasis or hypomotility—food taken many hours before or on the previous day may be seen in the vomitus.

The ***consistency*** depends largely upon its contents:

(*a*) *Fluid:* Thin watery vomitus may occur after an alcoholic debauch, in chronic gastritis, and, after having consumed large quantities of water which an irritable stomach may expel. If the vomitus is of *alkaline* reaction, it indicates the presence of a large amount of saliva and is often found when prolonged nausea has preceded the act of vomiting. *Acid vomitus* occurs in gastric hypersecretion and in acid fermentation, and may be found in peptic ulcer, gastric crises of tabes, hysteria, Graves' disease and migraine. *"Rice water" vomitus* is found in cholera.

(*b*) *Semisolid Vomitus:* This consists of undigested or recently ingested food; it occurs in gastric irritation, overfeeding, swallowing of nauseating food, seasickness (after a full meal), vertigo, etc.

(*c*) *Thick Tenacious Mucous Vomitus:* This is a symptom in acute or chronic gastritis.

Color: Green or yellowish vomitus is usually caused by bile in the stomach. It may be found after violent vomiting with retching, and in patulous pylorus. Vomiting of grass green bile in small amounts and unattended by retching is of frequent occurrence in peritonitis with intestinal obstruction. Yellow, blue, black, red (not blood) and other colored vomitus may be caused by the ingestion of coloring matter contained in food, candy, drinks or other substances

Red Vomitus—Hematemesis (vomiting of blood): The quantity of blood in the vomitus may vary from a few streaks or "pin points" to a quantity so large that the entire vomitus may consist of pure blood. Bright red blood indicates that the blood is fresh. Dark red, brown, black and coffee-ground color indicates that the blood has remained in the stomach for some time and undergone digestion. Hematemesis may be of *extragastric* or of *gastric origin*

Hematemesis of extragastric origin may be due to. 1. *The swallowing of blood* from a wound in or about the mouth, *i. e.*, the lips, gums, tongue, tonsils, rhinopharynx, after or during epistaxis, also from varicosities in the esophagus and injury of the esophagus by the swallowing of hard or sharp substances.

2. *Blood Dyscrasias:* Purpura, hemophilia, scurvy, severe primary and secondary anemia, leukemia, hemolytic jaundice, cholemia and at times Hodgkin's disease, acute fevers such as severe malaria, typhus, epidemic influenza, relapsing fever, yellow fever (black vomit), smallpox, dengue, chronic nephritis,

(*e*) ***Gastrectasis:*** Large quantities of fluid and particles of food are vomited at considerable intervals.

(*f*) ***Gastric Hyperesthesia:*** Vomiting occurs as soon as food or drink is swallowed.

(*g*) ***Hyperacidity and Hypersecretion:*** These may cause hyperesthesia with instant vomiting after taking food

(*h*) ***Asiatic Cholera:*** Asiatic cholera causes gastric irritation, frequent vomiting of a rice-colored material; it is unattended by nausea and is not followed by relief

(*i*) ***External pressure upon the stomach,*** such as pressure by a large liver or gallbladder, pericardial effusion or ascites may cause vomiting when the stomach is full.

II **Systemic Origin:** (*a*) ***Pulmonary Tuberculosis:*** Vomiting is caused by toxemia and occurs often after a paroxysm of cough

(*b*) ***Whooping cough*** and other forms of cough attended by strain may be followed by vomiting.

(*c*) ***Peritonitis*** causes vomiting of the gastric contents, bile and fecal matter.

(*d*) ***Disease or irritation of the bowel,*** *i e*, enterocolitis, appendicitis, colic, drastic purgation, etc., may cause vomiting.

(*e*) ***Acute obstruction*** of the bowels as in intussusception, volvulus, torsion, ileus and strangulated hernia causes vomiting *which gives no relief, and the* vomitus may become stercoraceous

(*f*) ***Biliary and renal colic,*** acute nephritis, pyelitis, cystitis and pancreatic disease may cause spontaneous vomiting.

(*g*) ***Addison's disease,*** and acute yellow atrophy *of the liver cause* characteristic vomiting.

(*h*) ***Toxins,*** poisons, uremia and eclampsia always cause vomiting.

III. **Nervous Origin:** *Vomiting of* central origin is usually not attended by nausea; it is of the projectile type, is not followed by relief and occurs independent of taking food.

(*a*) Tumor and abscess of the brain, meningitis, anemia and hyperemia of the brain, contusion and concussion of the brain, fracture of the skull; (*b*) seasickness, Ménière's disease, and migraine; (*c*) acute myelitis, disseminated sclerosis and paresis may be considered in this classification.

IV. **Reflex Vomiting:** This may be caused by: (*a*) Irritating and tickling of the pharynx and fauces, (*b*) persistent coughing; (*c*) attempt at dislodging viscid secretion from nasopharynx; (*d*) eyestrain; (*e*) revolting sights, (*f*) unpleasant odors; (*g*) sudden shock, nervousness, anticipation, anxiety or hysteria; (*h*) early pregnancy (morning sickness); (*i*) gastric crisis of tabes; (*j*) allergic manifestation; (*k*) heart disease (during the stage of decompensation), myocardial degeneration, pericarditis and angina pectoris Hiccough is a frequent complication in vomiting of reflex cardiac origin.

Persistent vomiting of Lydan is a form of reflex vomiting in which the attacks are recurrent without obvious cause, often associated with slight indigestion, constipation, fatigue, worry or disappointment. *The vomiting* is copious and continuous. Examination will reveal epigastric tenderness, retracted abdomen, hypersensitiveness and intolerance to light, sound and odors.

V. **Irritation of the Vomiting Center:** (*a*) *By drugs, i. e.,* apomorphine, morphine, digitalis; (*b*) *by toxemias*—nephritis, uremia, certain brain

any cause, in general peritonitis and in the presence of a gastrointestinal fistula.

Pus in the Vomitus: This may result from swallowing the contents of a retropharyngeal abscess, a peritonsillar abscess, or an esophageal abscess. The pus from empyema, pyopericardium, hepatic abscess, splenic or perirenal abscess may find its way into the stomach and be subsequently vomited. Phlegmonous gastritis and diphtheritic inflammation of the stomach wall may be primary causes of purulent vomiting.

Quantity, Odor and Reaction: These depend largely upon the quantity of food in the stomach, the kind of food, and the stage of digestion.

matter or the hearing of a revolting tale may cause nausea. It may occur also in diseases of the central nervous system, in neurasthenia and in hysteria.

Pain: *Cardialgia* is severe epigastric pain occurring in paroxysms. *Gastrodynia* is severe cramplike pain in the epigastric region. *Gastralgia* denotes pain in the stomach. *Pseudoangina pectoris* is severe pain in the epigastrium and lower sternal region, often referred to the shoulders. This may be caused by duodenal ulcer and adhesions in the right upper abdominal quadrant.

Epigastric pain, sharp or dull, constant or paroxysmal in relation to taking food or independent of it is an al-

Differential Diagnosis, Pulmonary and Gastric Hemorrhage

Hemoptysis	Hematemesis
1 Evidence of preexisting pulmonary disease	1 Referable to the throat, stomach, liver, heart or develops in females near the time of puberty.
2. Preceded by thoracic oppressions and a saline taste.	2 Preceded by giddiness, faintness or nausea
3. Blood ejected by coughing, or by clearing the throat, when hemorrhage is small	3. Blood ejected by vomiting or gagging
4 In profuse hemorrhage and when ejected immediately blood is arterial in color.	4. Blood of gastric origin is dark, as a rule, blood of pharyngeal origin, bright red.
5. Alkaline reaction	5 Gastric blood acid, pharyngeal blood alkaline, in reaction
6. Blood mixed with particles of mucopus	6. May contain undigested food
7 A pronounced beaded froth	7. Froth less marked
8 Microscopically, tubercle bacilli or other organism and possibly fibers of elastic tissue	8 Microscopically, sarcinae ventriculi, starch granules, particles of food, and in the case of carcinoma, large nonmotile bacilli (Oppler-Boas) and rarely carcinomatous tissue.

The blood from hemoptysis may be swallowed and later vomited.

Symptoms Preceding Emesis: It is important to note whether vomiting is preceded by nausea or pain.

Nausea: Nausea usually precedes vomiting of gastric origin, though it may occur in eyestrain (astigmatism), seasickness, early pregnancy, and in some the sight or odor of obnoxious

most constant symptom in most of the gastric disorders. At times the pain may be referred to distant parts of the body (See: p. 74).

There are also conditions other than gastric disease that may cause epigastric pain and should be differentiated from it.

Weil's disease, portal obstruction, atrophic cirrhosis, yellow atrophy of the liver, passive congestion of the liver, mitral stenosis and cardiac decompensation may cause hematemesis. The diagnosis of the underlying cause of hematemesis may be made by considering the general symptomatology and physical and laboratory findings

3. *Vicarious Menstruation*· Hematemesis occurring periodically either with the menstrual period or instead of it, has been described by various clinicians A thorough examination of the patient should be made before a definite diagnosis of vicarious menstruation is made, because this usually occurs during the "ulcer age" of the individual.

4. *Hematemesis* may also occur in acute pancreatitis, in appendicitis, in cholecystitis, in mesenteric embolism or thrombosis and as a result of focal infection within the abdominal cavity After abdominal operations hematemesis may appear as a grave complication

5. *Hemoptysis or bloodstained expectoration* may be swallowed and subsequently vomited.

Hematemesis of Gastric Origin· 1. *Gastric Ulcer* The vomitus is usually bright red and copious. It may be preceded by sharp epigastric pain. At times hematemesis may occur without any premonitory signs of indigestion or pain and the gastric hemorrhage may appear as suddenly as a thunderbolt from a clear sky. Duodenal ulcer may cause hematemesis several hours after the ingestion of food. It is usually accompanied by melena, and presents other signs of duodenal ulcer.

2. *Gastric Carcinoma*. The blood usually shows some digestive change and may be of dark brown or coffee-ground color, indicating that the blood had remained in the stomach for some time. The quantity of blood may be little or copious. Vomiting occurs frequently and the amount is usually small.

3. *Miliary Aneurysms and Varicosities of the Stomach and Esophagus:* The bleeding is usually profuse. It occurs, as a rule, in persons past 60 with arteriosclerotic changes in other vessels. The blood is bright red and is brought up in small clots.

4. *Injury* to the epigastric area by blows or kicks may be followed by hematemesis. The quantity is variable and is accompanied by severe epigastric pain and rigidity of the recti abdominis muscles.

5. *Poisons:* Corrosives, *i e*, arsenic, bichloride of mercury, phosphorus, strong acids and alkalies cause bloody vomitus which may be of small quantities, and is oft repeated. Vomiting is accompanied by retching and pain. The vomitus contains not only blood, but also mucus and shreds.

6 *Straining or Retching* during the act of vomiting may cause streaks of blood mixed with the vomitus.

Contents: The contents of the vomitus may be food, mucus, saliva, gastric juice, blood and parasites, *i e.*, roundworms, threadworms, trichinae, anchylostomiasis duodenale, blood flukes, tapeworm and fragments of echinococcus cysts

Fecal Vomiting (stercoraceous): Actual fecal masses are seldom seen in the vomitus. The intestinal contents when vomited consist of a blackish or brownish fluid of a distinctly fecal odor. Fecal vomiting is preceded first by vomitus of the gastric contents, then by bile (duodenal contents) and finally by the lower intestinal contents. Stercoraceous vomiting occurs in intestinal obstruction from

abdominal muscles, epigastric hernia, hydronephrosis, carcinoma of the transverse colon, adhesive pericarditis, pericardial effusion, large pleural effusion, cardiac dilatation, aneurysm of the thoracic aorta, angina pectoris, aortitis, and lead poisoning. Epigastric pain is often present in sudden emotions, mountain climbing and severe exhaustion.

Cardiac Palpitation of Gastric Origin: This may occur as a result of overeating, gastric flatulence, the ingestion of improper food, overindulgence in alcohol and tobacco and in neurotic individuals when eating while under stress, fear or excitement.

Diseases of the Stomach

Many of the diseases of the stomach cannot be accurately diagnosed by the evaluation of the history, symptomatology, and the physical signs. For accurate diagnosis it often becomes necessary to examine the stomach contents, to have an x-ray study and, at times, a gastroscopic study.

Gastralgia (Gastrodynia, Neuralgia of the Stomach)

Gastralgia is a condition of the stomach characterized by severe paroxysmal epigastric pain, unassociated with any definite gastric lesion It may be caused by overwork and anemia, or by such dietetic errors as may produce acute gastritis. This condition is usually found in people of a sensitive and nervous temperament Gastralgic pain is often associated with gastric cancer and ulcer; it is also found in locomotor ataxia and nervous dyspepsia with hyperacidity.

Symptomatology and Diagnosis: Paroxysms of severe pain in the epigastrium, usually radiating to the back, occur when the stomach is empty. Relief may be had by pressure upon the painful area, and the ingestion of warm stimulating drinks and food.

Differential Diagnosis: Simple gastralgia should be differentiated from the following conditions:

Gastric Ulcer: Pressure in the epigastrium causes pain; hyperchlorhydria is always present; vomiting of blood often occurs, and the ingestion of food may increase pain

Carcinoma: Anemia, often emaciation, almost continuous pain, which increases after taking food, loss of appetite, vomiting at times with coffee-ground material and an absence of hydrochloric acid with the presence of fatty acids, and an epigastric mass are strong diagnostic features.

Angina Pectoris: Pain usually comes on after exertion, as a rule it is over the lower part of the sternum or precordium and radiates to one or both shoulders, and down the left arm. During the attack the patient is oppressed by a sense of impending death

Gastric Crisis of Locomotor Ataxia: This sometimes simulates gastralgia, but the history of syphilis and other tabetic signs would lead one to suspect this condition.

Caries of a Vertebra, Aneurysm of the Thoracic Aorta, Pericarditis, Dietl's Crisis; Renal Colic, Cholelithiasis, Acute Pancreatitis. These are conditions that should be borne in mind when one attempts to diagnose gastralgia. The history of the patient, the physical signs and x-ray study will often help in arriving at a correct diagnosis

Peptic Ulcer (Gastric Ulcer and Duodenal Ulcer)

Definition: A peptic ulcer is a round perforating ulcer occurring in the stom-

Differential Diagnosis, Inflammation, Neuralgia and Colicky Pain in Abdominal Region

Pain	Inflammation	Neuralgia	Colic
Type and radiation.	Dull aching, and if the inflammation is acute and engorgement of the vessels is excessive, the pain also tends to radiate from the inflamed area outward toward the periphery	Sharp, acute, generally radiates along the course of a nerve, as in neuralgia of the tenth dorsal nerve, in which the pain radiates around from the tenth interspace to the area of distribution on *the abdominal wall*	Sharp, acute and agonizing; the pain of a colic radiates in different directions depending upon the location of the colic, for instance, in gallstone colic the pain radiates around to *the back underneath* the scapula of the same side
Pressure	Increases the pain	Is excessively tender The slightest pressure produces an excruciating pain Pain can also be produced by pressure upon the nerve trunks and this pain radiates *along the terminal* branches	Eased by pressure; as in cases of gallstone colic, the patient seeks ease by doubling up and making pressure against the abdominal wall
Duration	Constant	Intermits, but intermission is not sudden and acute	Stops suddenly, but the soreness persists for a short time
History	Generally has not had a previous attack	May not have had previous attack	Generally a history of previous attack

Epigastric pain referred to the left of the spinal column accompanied by epigastric tenderness and aggravated soon after taking food, which is relieved by vomiting is suspicious of *gastric ulcer*

Epigastric pain which occurs two or three hours after taking food but is *relieved immediately after taking food* or alkalies is significant of *duodenal ulcer*.

Epigastric pain which is nearly constant and is not *relieved by alkalies*, and is accompanied by tenderness and the presence of a mass in the epigastrium, is suspicious of *carcinoma*. During the early stages of carcinoma, a tumor may not be palpable.

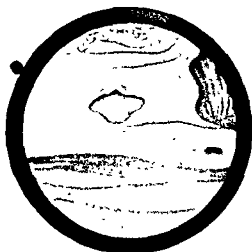
Epigastric pain accompanied by a burning sensation (heartburn) which occurs after taking rich spicy foods, acids, alcoholic beverages or after the excessive use of tobacco is indicative of *acute gastritis*.

Epigastric pain accompanied by overdistention of the stomach with a sense of fullness in the epigastrium often with the sensation of a "lump in the throat" is indicative of *gastric fermentation*

Epigastric pain and tenderness occurring in paroxysms and referred to the right shoulder is significant of *gallbladder disease*.

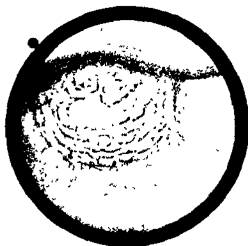
Epigastric pain, *occurring* in paroxysms which are acute and sharp, often accompanied by collapse and tenderness above the umbilicus, and associated with a slow pulse is significant of *pancreatic disease*.

Epigastric pain may be a symptom in Dietl's crisis, gastric crisis, acute intestinal obstruction, necrosis of a vertebra, intercostal neuralgia, myalgia of



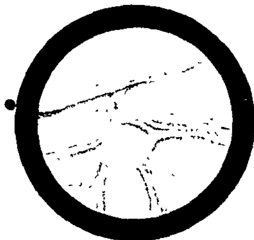
GASTRIC ULCER

A definite ulcer involving the lesser curvature, the edges of which are undermined The ulcer shows yellowish discoloration The dark area to the right is the pyloric antrum Just underneath, a small pigment fleck (dark brown) is noted Above, small air-containing blebs of mucus and submucous hemorrhagic areas are seen



CALLOUS GASTRIC ULCER

A large callous ulcer involving the lesser curvature is seen It is of the deep penetrating type leading to the pancreas



GASTRIC ULCER SCAR

A scar resulting from a gastric ulcer is seen on the anterior wall of the stomach

ach, duodenum and in other parts of the gastrointestinal tract to which acid gastric juice has access. It most frequently occurs in young males and females, though a sharp age limit cannot be set.



Fig. 5—Perforating ulcer of the stomach. (Courtesy of Leon Solis-Cohen)

Etiology: The actual etiologic factor of peptic ulcer is not clear. Many predisposing factors may be operative in its production, *i. e.*, hyperacidity, irritating foods, excessive smoking of tobacco, anemia, focal infections, body burns, intracranial lesions, and trauma. Why only a certain percentage of people and not all of those who are predisposed to one or several of the above named causes should develop ulcer, has not, as yet, been explained.

Symptomatology and Diagnosis: *Pain is the most constant symptom. It* may be sharp and lancinating, and is felt in the precordium, radiating posteriorly at times to the left of the spine on a level with the tenth rib. The pain may be dull and gnawing, or it may only cause a sense of uneasiness or a burning sensation in the epigastrium. The severity of the pain depends upon the associated inflammation of the gastric mucosa.

*(a) **Time of Pain:*** In gastric ulcer the pain may occur in from 10 or 15 minutes, to two or three hours after taking food. At times the pain may be aggravated by food, and at other times food will relieve pain, the relief only lasting until such time as an excess of hydrochloric acid is again secreted into the stomach and causes irritation. The time of pain often depends upon the location of the lesion. An ulcer close to the cardia may cause pain soon after eating, while one near the pylorus may not cause pain until an hour or two after the taking of food. Duodenal ulcers usually give rise to pain from two to four hours after eating.

*(b) **Epigastric Tenderness:*** This and slight rigidity may be elicited in the great majority of cases. A tender point to the left of the spine in the tenth or eleventh intercostal spaces is frequently found.



Fig. 6—Duodenal ulcer. (Courtesy of Leon Solis-Cohen.)

*(c) **Indigestion:*** This is another constant symptom. It is usually because of pain and indigestion, that the sufferer

Differential Table Between Gastric Ulcer and Gastralgia

Gastric Ulcer	Gastralgia
History unimportant.	History of neurasthenia, neuralgia and hysteria the rule.
Most frequent from 15 to 35 years of age.	Most frequent before or near the menopause (in the female).
The paroxysms of pain usually come on at a definite period after eating	Paroxysms more frequent when stomach is empty and show less periodicity.
Eating relieves pain for a short period	Eating usually brings relief.
Position of patient may relieve pain	No decided relief.
Tenderness on pressure over a certain limited area in the epigastrium.	Tender spot absent. General hyperesthesia of the skin of epigastrium often present.
Pressure usually aggravates and only occasionally relieves patient during paroxysms of pain—not during the intervals between seizures.	Pressure almost always relieves the pain.
In the intervals gastric disturbances, more or less severe, are present.	In the intervals between attacks no gastric disturbances present, as a rule.
Hematemesis present in nearly one-half of the cases.	Hematemesis absent.
General health often much impaired, particularly late in the affection	General health less affected than in ulcer.
Physical signs of a mass may be present.	Signs of tumor always absent.
Dilatation may coexist, in the late stage.	Dilatation not present.
Hyperacidity of gastric juice usually present.	Hyperacidity present only in certain forms.
Improvement follows rest and regulation of diet	Regulation of diet has no effect.

seeks a physician's advice Fullness after meals, eructation, and pyrosis are often complained of for many years before a diagnosis of ulcer is made.

(*d*) ***Vomiting:*** Nausea and vomiting may occur at infrequent intervals. The vomitus may contain large quantities of acid material and food in various stages of digestion depending upon the time elapsed between the ingestion of food and its expulsion through the mouth

(*e*) ***Hematemesis:*** This occurs in a large proportion of cases Sometimes a frank hemorrhage, at other times only a small quantity of blood mixed with food, and occasionally only occult blood may be found In duodenal ulcer there may be hematemesis with melena, or occult blood in the vomitus and feces. Hematemesis may be the first sign of a peptic ulcer, no other symptoms may be complained of by the patient preceding the bleeding.

(*f*) ***Anemia:*** This may occur because of malnutrition, hematemesis and because the food is not being properly assimilated or is vomited. Persistent bleeding, no matter how small the quantity of blood lost each day, may cause grave anemia.

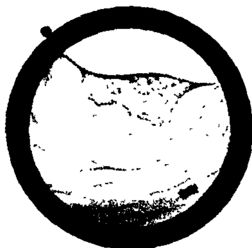
Gastric Carcinoma

This usually occurs in persons past 40 years. Among the predisposing causes are age, mechanical irritation—such as ulcer or hot fluids or irritating substances—and probably heredity. A carcinoma may affect the cardiac end of the stomach, the greater or lesser



ANNULAR CARCINOMA OF THE PYLORUS

A large ulcerative extensive carcinoma is seen involving the pylorus in circular fashion



ULCERATIVE CANCER OF THE PYLORUS

Above is seen the large ulcerative carcinoma infiltrating the posterior wall Below to the right, is seen a small carcinomatous ulcer surrounded by healthy mucosa.



GASTRO-ENTEROSTOMY STOMA

At the right, the gastro-enterostomy stoma is seen, the edges well defined by folds of the mucosa At the left the normal appearing pylorus is seen

curvatures or the pylorus. In some instances the entire stomach may be infiltrated giving it a leather bottle appearance.

Symptomatology and Diagnosis: (*a*) ***Pain:*** Gastric pain is usually constant, at times it may come on soon after taking food, or not until one, two or more hours later. The closer the lesion is to the cardia the earlier in the digestive period does the pain occur The pain may be burning, dragging or boring associated with a sense of suffocation after meals. The appetite is poor, though some patients retain their appetite until late in the disease.

(*d*) ***Loss of Weight:*** Progressive loss of weight is a constant feature. At first, weight is lost slowly and as the disease progresses, emaciation occurs *rapidly*

(*e*) ***Anemia and Cachexia:*** These occur as the disease progresses The blood picture is that of secondary anemia



Fig 7—The arrow points to a neoplasm which involves the pyloric portion of the stomach

in character, and continuous or paroxysmal. In some instances pain does not occur until after the carcinoma has become moderately far advanced

(*b*) ***Vomiting:*** This is an early symptom and is usually preceded by nausea The vomitus is often blood-stained, having a coffee-ground appearance, particularly so if the food and blood have remained in the stomach for some time.

(*c*) ***Dyspepsia:*** Indigestion, epigastric distress, the sensation of fullness and of a lump behind the sternum often

Gastric Analysis: This will reveal an absence of free hydrochloric acid even after the histamine test and the presence of fatty acids. (For further detail, see p. 1028)

Physical Examination: This reveals usually a pale, pasty-looking individual who gives evidence of having lost weight; a mass may be palpated in the epigastrium which, as a *rule*, is *not* very tender to touch The size of the palpable mass depends upon the stage of the disease Metastasis to the lymph glands and to other organs may occur.

Acute Gastritis (Simple Gastritis, Acute Dyspepsia, Acute Gastric Catarrh)

Acute gastritis is an acute disturbance of the stomach, occurring as a result of indiscretion in diet, either quantitative or qualitative. The ingestion of alcohol, spiced foods, pastries and other indigestible articles, overeating or eating when one is exhausted or in a great hurry or under some emotional strain, are among the predisposing features.

Symptoms: These are epigastric distress, fullness, sensation of being bloated after meals, nausea and occasional vomiting, headache, at times diarrhea alternating with constipation, and abdominal colic. Vomiting usually gives relief

Physical examination is practically negative.

Acute gastritis may be caused by some definite inflammatory condition of the gastric mucosa.

Thomas McCrae describes *suppurative gastritis, toxic gastritis, diphtheritic* or *membranous gastritis* and *mycotic* and *parasitic gastritis* as follows:

Suppurative Gastritis: This is characterized by epigastric pain, high fever, vomiting, dry tongue, and other symptoms of acute infection. Jaundice is sometimes present.

Toxic Gastritis: This is characterized by intense pain in the mouth, throat and stomach, difficulty in swallowing, salivation, and more or less constant vomiting; sometimes the mucous membranes of the stomach and blood may be found in the vomitus; the abdomen is usually distended and tender to touch. This condition is caused by the ingestion of poisons such as carbolic acid, bichloride of mercury, arsenic, phosphorus, oxalic acid, etc

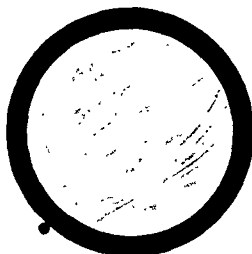
Diphtheritic or Membranous Gastritis: This sometimes occurs in diphtheria; however, membranous gastritis may be found in severe toxic fevers such as typhus or typhoid fever, smallpox, pneumonia, pyemia and the membranous gastritis of childhood. This condition is diagnosed by the occurrence of membranes in the vomitus, pain, fever and symptoms of the associated underlying diseases.

Mycotic and Parasitic Gastritis: Various fungi and bacilli may often reside in the gastric mucosa and set up an acute or chronic inflammation, the specific diagnosis of which can be made only when the organisms are recovered in the vomitus.

Chronic Gastritis (Chronic Catarrh of the Stomach, Chronic Dyspepsia)

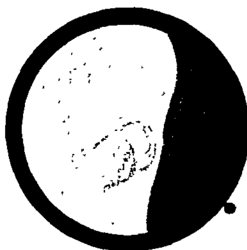
By chronic gastritis is meant a chronic catarrhal inflammation of the gastric mucous membrane, associated with qualitative and quantitative changes in the gastric juice, the formation of large quantities of mucus with alterations in the size of the stomach and the tonus of its walls This may be caused by improper, indigestible food, or by food that is too hot or too highly seasoned, the abuse of alcohol, tobacco and ice water; by focal infection, such as chronic appendicitis, infected teeth, tonsils or infected sinuses; chronic diseases, such as diabetes, nephritis, anemia, tuberculosis, etc ; also by organic inflammatory diseases of the stomach, such as carcinoma or ulcer

Symptomatology and Diagnosis: This condition is gradual in its onset, which is characterized by occasional attacks of indigestion and the inability to digest certain foods; nausea, and occasional vomiting after a full meal. As the



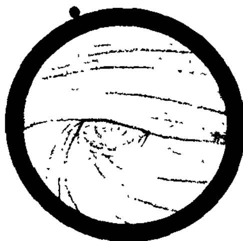
CHRONIC GASTRITIS.

The mucosa of the fundus portion of the stomach evidences a chronic catarrh Red spots are seen near the small vascular endings (probably hemorrhagic). This is probably a case of beginning gastric atrophy.



HYPERTROPHIC POLYPOID GASTRITIS

The posterior wall of the stomach evidences a hypertrophic gastritis with polypi which are rather prominent due to the associated swelling of the mucosa



CARCINOMA OF THE PYLORUS

A large carcinoma involving the pylorus is seen just below the lesser curvature level.

Differential Diagnosis, Chronic Gastritis, Gastric Ulcer and Gastric Carcinoma

Chronic Gastritis	Gastric Ulcer	Gastric Carcinoma
Not confined to any age More common in middle-aged or elderly people.	May occur in middle-aged persons, but is most frequent in young adults	Most common in elderly people; rarely occurs in persons under 40 years of age.
Pain in the epigastrium somewhat aggravated by food; soreness is also present. Both are constant, although comparatively slight	Pain in the epigastrium much aggravated by food; subsides when this is digested; paroxysms of pain not lancinating; strictly localized soreness to touch in epigastrium; sometimes a painful spot over lower dorsal vertebrae Intermissions in the pain are frequent.	Pain frequently of a radiating kind, often paroxysmal, not infrequently severe and lancinating, but not of necessity associated with soreness Little or not at all affected by food. Pain rarely remits, never intermits for any considerable time.
Symptoms of indigestion marked.	Symptoms of indigestion sometimes slight. Heartburn and pain frequent.	Symptoms of indigestion marked. Anorexia; extreme acidity of stomach.
Sometimes vomiting.	Vomiting may be present or absent.	Vomiting a very frequent symptom
No hemorrhage, or but trifling hemorrhage; at most blood streaks in vomited matter	Abundant hemorrhage from the stomach common Stools may contain blood (tarry).	Hemorrhage not very abundant, but frequently occasioning coffee-ground vomit.
Bowels constipated.	Bowels usually constipated Intermittent occult blood in stools.	Bowels obstinately constipated Occult blood in feces continuously.
No fever.	No fever.	Attacks of slight fever occur; temperature often subnormal.
Acid taken with meals does not increase pain.	Acids taken increase pain.	Acid taken does not increase pain.
Not much emaciation; no cachectic appearance.	Frequently extreme pallor and debility, especially if preceded by anemia	Progressive loss of flesh, and cachexia; enlarged lymphatic glands
Disease may be relieved or cured; is often of very long duration	Duration uncertain; may get well, may run on rapidly to perforation; or may last for years.	Average duration one year; may be shorter, but seldom longer.
No tumor.	Rarely a tumor.	Generally a tumor.
Contents of stomach almost always contains free hydrochloric acid.	Hydrochloric acid in excess in contents of stomach.	No hydrochloric acid in contents of stomach.
No lactic or fatty acids after the rigid Boas test meal	No lactic or fatty acids after the rigid Boas test meal	Lactic acid present *after* Boas test meal.
Slight motor disturbance.	Motor function fair.	Early marked disturbance
No dropsy.	No dropsy.	Edema of ankles common.

quantity of fluid present in the stomach If the stomach is distended with gas it may easily be outlined If it is partially filled with fluid or solid material, its exact boundary is not easily mapped out By *auscultatory percussion* or a vibrating tuning fork, the boundaries of the stomach may at times be outlined. The most reliable method for determining the size of the stomach is an x-ray examination

Hypertrophic Stenosis of the Pylorus

Nonmalignant thickening due to hypertrophy of the muscular and mucous coats of the pylorus may be congenital or acquired.

Congenital Stenosis: This is a condition seen in very young infants, it is usually associated with pylorospasm and is recognized by frequent vomiting, rapid emaciation and visible peristalsis. Peristalsis may be enhanced by irritating or tickling the skin

Acquired Stenosis: This usually occurs in the adult and may be benign or malignant The symptoms of nonmalignant and malignant pyloric stenosis are similar, *i e*, vomiting, rapid emaciation, etc

Gastroptosis and Enteroptosis (Glénard's Disease)

Glénard's disease is a downward displacement of the stomach and intestines This is found most frequently among women and may be caused by tight lacing or repeated pregnancies, it is also seen among persons who undergo muscular strain, rapid emaciation and malnutrition As a general rule, when there is ptosis of the stomach, displacement downward of the spleen, kidneys, liver and colon accompany it.

Symptomatology and Diagnosis: Examination usually reveals a nervous, rather emaciated person who presents symptoms of nervous dyspepsia, flatulence, constipation, colicky pains and neurasthenic manifestations The lower abdomen appears pendulous and unusually distended, the concave lines of the upper abdomen are greatly exaggerated The general posture of the patient resembles a question mark Tympany may be elicited in the lower abdomen An x-ray study would indicate the true character of the conditions

Neurosis of the Stomach (Nervous Indigestion)

Under this heading may be considered certain functional disorders of the stomach which are characterized by recurrent attacks of gastric disturbance followed by intervals of complete freedom from symptoms These conditions usually occur in emotional and highly neurotic individuals and may be ushered in by mental stress, grief, intensive joy, startling news, depression or great anxiety It may also occur reflexly because of disease of the gallbladder, bile ducts, appendix, pancreas, colon and exophthalmic goiter A diagnosis of gastric neurosis should be withheld until exhaustive studies have failed to discover an organic lesion or any other definite cause for the digestive disturbances.

Symptomatology and Diagnosis Among the prominent symptoms are anorexia alternating with excessive appetite, eructation of gas, epigastric distress, heartburn and occasional regurgitation of food, with or without occasional vomiting. The gastric content is usually normal and the x-ray examination reveals nothing abnormal

Physical Examination: This will reveal a nervous individual, who may either be emaciated or the picture of

Differential Diagnosis, Gall-Duct Disease, Gastric Ulcer and Pyloric S

Symptoms	Gall-Duct Disease	Gastric Ulcer	Pyloric
Pain type.	Generally some constant soreness in cholangitis, then, as the duct becomes blocked, the pain is paroxysmal with a gradual disappearance, only a soreness remaining The pain may be referred to the area of the fourth costal cartilage on the left side. Long intervals from pain may be present.		lieved by Attacks ge cur at sh vals
Relationship to the ingestion of food.	None except in cases of inflammation of the duct (common), when it seems that intestinal peristalsis *may set up an associated* peristalsis in the duct.	Follows immediately upon or a short time after the ingestion of food, depending upon whether *the ulcer is at the cardia* or the pylorus Eased by local analgesics	Follows tw hours aft gestion of f
Tenderness	Slight tenderness in epigastrium, then over the gallbladder and liver area, as the duct becomes occluded and the gallbladder and liver distended.	Present in a circumscribed area Area is constant, and is generally located in the epigastrium immediately below the ensiform cartilage	In epigastriu
Jaundice	Present (usually).	Absent.	Absent
Nausea and vomiting	*Generally present, constant* Bile is present when the duct is blocked. May ease the pain.	*Generally occurs* Some blood in it at times, the pain is generally eased by it Bile present.	Frequent; ea pain, no bile
Temperature.	May have a Charcot's intermittent fever, but generally no rise in gallstone colic and only a slight rise in cholangitis	*No rise*	*No rise*
Pulse	Generally slight increase	Slight increase.	Slight increase
Urine.	Bile present.	No bile	No bile
Position of election	Gallstone colic, patient is doubled up, with knees flexed on abdomen, body bent forward, and pillow or hands *often placed against* abdomen. Patient often lies on his face	Back	Any position
Effect of movement	Patient is very restless, constant movement	Restless	Very restless, pressed ti against the ab
Application of cold or heat	Same as in gallbladder.	Cold eases	Cold increases, eases.

tration of histamine, may be found in cases of advanced atrophy of the gastric mucosa, in pernicious anemia and occasionally in other anemias, locomotor ataxia, carcinoma of the stomach, and at times in otherwise apparently normal individuals.

III. **Sensory Neurosis:** This is characterized by the following symptoms:

(*a*) *Hyperesthesia* is a supersensitiveness of the gastric mucosa in which the patient complains of fullness, burning, gastric distress, often before the meal is completed and at times when the stomach is empty.

(*b*) *Gastralgia* may occur as a manifestation of gastric neurosis or as the result of organic disease.

(*c*) *Anomalous sense of hunger* may occur, *i. e.*, the patient may be constantly hungry, may have no appetite at all, or may have a craving for unusual foods or other articles (SEE: p. 89).

The Pancreas

Physical Examination of the Pancreas

Physical examination of the pancreas is not satisfactory because of its anatomic position. The presence of a tumor, carcinoma, suppurative pancreatitis, acute hemorrhagic pancreatitis, or a cyst of the pancreas can only be surmised by the sense of resistance and pain elicited by deep palpation over the abdomen midway between the umbilicus and the xiphoid cartilage The close proximity of the head of the pancreas to the portal veins, the inferior vena cava and the ductus communis choledochus are of clinical importance

The pancreas is a gland possessing an internal and external secretion. The islands of Langerhans are the glands of internal secretion, which secrete insulin. Disease of these glands is responsible for disturbed carbohydrate metabolism and results in either hyperinsulinism (hypoglycemia) or in hypoinsulinism (hyperglycemia) as in diabetes mellitus (SEE · p 798).

The external secretion of the pancreas is represented by the enzymes Disease of the pancreas proper may alter the quality and quantity of the pancreatic enzymes and interfere with digestion primarily of fat, protein material and possibly nuclear material.

Diseases of the Pancreas

Pancreatitis

Acute Pancreatitis: This is an acute inflammatory disease of the pancreas characterized by necrosis, gangrene or suppuration of portions of the gland and usually is associated with hemorrhage.

Symptomatology and Diagnosis: An attack of acute pancreatitis is ushered in by sudden intense pain in the epigastrium followed by severe vomiting and belching of gas, and is frequently accompanied by hiccoughs, and symptoms of profound collapse. The pain is usually continuous with periodic exacerbations, and radiates to the back and to the left hypochondrium. At times it may be referred to the lower abdomen. The abdomen is usually distended. There is an area of rigidity and tenderness above the umbilicus Vomiting at frequent intervals of stomach contents and of bile may accompany the distention. Flatus may be passed, though the abdomen is silent. Constipation is marked. The pulse is

health; all other findings are negative except that hyperperistalsis may be present.

Neurosis of the stomach may be of three varieties (I) Motor neurosis, (II) secretory neurosis and (III) sensory neurosis. These may occur individually or collectively, and are found in nervous hypersensitive individuals whose symptoms may often simulate organic disease.

I. Motor Neurosis: This is characterized by.

(*a*) *Hypermotility* is manifested by an increase in the normal motor activity of the stomach and pyloric spasm.

(*b*) *Peristaltic unrest* exhibits peristaltic movements of the stomach and bowel soon after eating accompanied by gurgling and borborygmi.

(*c*) *Eructation* causes continuous or paroxysmal belching either of gas engendered in the stomach or of swallowed air. Air swallowing is a fairly common phenomena among nervous individuals.

(*d*) *Nervous vomiting* may occur at any time and even without provocation, it is not associated with nausea or pain, nervous vomiting when persistent may result in acidosis or alkalosis.

(*e*) *Rumination (merycismus)*, regurgitation of food which is chewed again and swallowed, occurs frequently

(*f*) *Cardiospasm* is characterized by pain on swallowing food and is caused by spasmodic contraction of the cardiac orifice; it also produces a sound as the food goes down This condition is found in air swallowers, hysterical and neurasthenic individuals, and also in tetanus

(*g*) *Pyloric spasm* is usually secondary to hyperacidity, hyperperistalsis, and ingestion of irritating foods

(*h*) *Atony of the stomach* itself may be found in neurotic individuals who abuse their stomachs by improper food or feeding or it may result from organic disease of the stomach.

(*i*) *In insufficiency or incontinence of the pylorus,* the pylorus is gaping and permits the stomach content to pass into the duodenum without any hindrance. It also allows regurgitation from the duodenum into the stomach.

(*j*) *Insufficiency of the cardia* causes a gaping of the cardiac orifice which permits eructation of food; this is most noticeable on change of posture or when pressure is made against the stomach This is also often observed in healthy infants when promiscuously handled after feeding.

II. Secretory Neurosis: This causes the following conditions·

(*a*) *Hyperacidity and hyperchlorhydria* is characterized by an increase in the amount of gastric juice and hydrochloric acid. It occurs in many gastric disorders of nervous origin, also in ulcer and acute gastritis.

(*b*) In *hypersecretion,* the gastric juice is increased in quantity, this may occur continuously or in paroxysms, often depending upon the kind of stimulus and the state of excitability of the individual.

(*c*) *Hypoacidity or anacidity-achylia gastrica nervosa* is characterized by a diminished amount of gastric juice which contains the normal gastric enzymes and does not interfere with the emptying time of the stomach. This may occur in nervous conditions and in such cases a test meal containing meat or the hypodermic injection of a minute quantity of histamine will increase the quantity of HCl in the gastric juice. The persistent absence of HCl and enzymes in the juice after a meat meal, or after the adminis-

Differential Diagnosis, Disease of the Pancreas, Renal Colic, Appendicitis

Symptoms	Pancreatitis	Renal Colic	Appendicitis
Pain, type	[illegible]	[illegible] may be a long period of freedom between individual attacks.	May, in case of colic, be of sudden onset. Finally is localized to the right inguinal fossa At first, because of the localization of the appendix pain in the epigastrium, it may be confused with cholecystitis In some cases, gallstone colic may be confused with appendiceal colic
Relationship to the ingestion of food	No special relation in the acute variety, but in chronic is made worse several hours after the ingestion of food.	No relationship.	May follow four to eight hours after taking food Rather common during the night
Tenderness	Epigastric (low).	Over the kidney region in the loin.	Over McBurney's point
Jaundice	Slight amount may be present.	Absent.	Absent.
Nausea and vomiting	Present, and as a rule, persistent. Bile generally present	Not so common.	Nearly always present.
Temperature	Rise or, if the shock be too great, a fall	No rise.	Rise, if the severity of the disease increases, the temperature continues to rise and may assume a septic type if abscess formation results.
Pulse	Very rapid, or very slow	Generally rapid	Increased in rapidity
Urine	Occasional glycosuria, no bile, urine and serum amylase and lipase high.	No bile, but blood and pus	Generally no bile
Position of election	On back	On back, with the knee of the affected side flexed on the abdomen	Dorsal, limbs drawn up, and thighs flexed on the abdomen
Effect of movement	Increases pain	Not much effect. Patient himself is very restless	Very quiet When peritoneum is involved, respiration is restricted
Application of heat or cold	Cold eases and heat increases	Heat eases	Cold eases Heat increases at times
Referred areas	To left hypochondrium and left abdomen	From affected kidney to epigastrium and along ureter to bladder.	Lower than in gallbladder or duct disease

slow and jaundice may be present. The stool, when passed, contains large quantities of fat and the urine may give a positive Cammidge reaction and an increased diastase index above 100 or 200.

Fitz's Rule: Acute pancreatitis is to be suspected when a previously healthy person or one suffering from occasional attacks of indigestion is suddenly seized with violent epigastric pain followed by vomiting and collapse and in the course of 24 hours by a circumscribed epigastric swelling which is tympanitic or resistant, a slight rise of temperature and the presence of fat necrosis

Suppurative Pancreatitis: This may be described as a diffuse suppuration of the pancreas, often associated with numerous small abscesses or one large abscess It may be: 1. Acute; 2. subacute, or 3. chronic.

Symptomatology and Diagnosis: 1. *Acute Suppurative Pancreatitis:* This starts abruptly with severe pain, vomiting, chills and hiccoughs, associated with a septic temperature. Pain is often referred to the left abdomen, slight jaundice and glycosuria may be present; constipation may be followed by fatty diarrhea. The serum and urine amylase is high. The condition is usually fatal.

2. *Subacute Suppurative Pancreatitis:* This is characterized by epigastric pain radiating toward the left, progressive emaciation, general weakness, copious fatty diarrhea, and septic temperature. This condition may last from three to four weeks, terminating in death.

3. *Chronic Suppurative Pancreatitis:* The symptoms are less severe but become progressively worse. It is characterized at first by mild epigastric pain, slight septic temperature, anorexia, anemia with gradual loss of strength, and at the terminal stage anasarca may supervene.

Hemorrhagic Pancreatitis: ***Symptoms:*** These are characterized by an acute onset of excruciating deep-seated epigastric pain occurring in paroxysms, nausea, retching and severe vomiting, constipation and severe collapse The vomitus may contain slimy mucus and dark blood. A slight rise of temperature, dyspnea, rapid and feeble pulse, delirium, jaundice, tympanitis, hiccoughs and cyanosis are usually present. Rigidity and tenderness above the umbilicus may be elicited.

Subacute Pancreatitis: This generally begins with slight epigastric pain, coming on several hours after meals. The pain steadily becomes worse until it resembles biliary colic. These paroxysms of pain may come on at frequent intervals, but gradually the intervals are lengthened and the severity of the pain lessens. When the disease becomes aggravated the intervals diminish and the paroxysms increase in length and severity. Pain is often referred to the lumbar region, at times to the lower abdomen and legs, resembling acute appendicitis or renal colic.

Symptomatology and Diagnosis: The paroxysms cause collapse. The patient has a grayish pallor and an anxious expression; the tongue is dry; retching and vomiting with blood, and in severe cases with fecal matter occur; the temperature is but little elevated; the pulse is slow and small. A mass may be palpable in the upper abdomen midway between the umbilicus and xiphoid Peritonitis may occur as a result of this condition. It is usually associated with gallbladder disease, peptic ulcer or duodenitis.

Differential Diagnosis of Pancreatitis and Intestinal Obstruction

Symptoms	Pancreatitis	Intestinal Obstruction
Pain	[illegible] ders	[illegible]
Jaundice	Present (often)	Absent
Pulse	Slow, except when shock is associated; then it is rapid and thready.	Gradually increasing in rapidity.
Tumor.	A gradual development of one in the epigastrium	Present, tympanitic over the region of the obstruction Rare in the epigastrium Not tender on pressure
Vomiting.	Present, generally persistent. Bile generally present, gradually becomes less frequent.	Vomiting, at first of stomach contents, then of bile and then of bowel contents
Fever	Present with chills	Absent at first
Distention.	Largely colonic, generally the tympany is marked, especially in epigastrium	May occur in any part of the bowel, always above the area of obstruction.
Free fluid in peritoneal cavity	Rapid development of.	Little if any free fluid
Shock.	Present	Absent.
Diarrhea	May or may not be present, excess of fat in stools	Obstipation
Hiccough	Present	Generally absent
Belching.	Present.	May be present
High enemata	Generally result in the passage of gas and fecal matter, and the reduction of the distention	Result in the passage of some fecal matter, and the cleansing of the large bowel, but with no lessening of the distention.
Urine	Glycosuria intermittently present	No bile, no sugar

Symptomatology and Diagnosis: The symptoms most frequently encountered in this condition are slight colicky paroxysmal pains referred either to the epigastrium or along the hypochondrium, vomiting, constipation or fatty diarrhea, jaundice and ascites (in the presence of large cyst); the diagnosis of this condition may be inferred when a large mass is found in the midabdomen above the umbilicus in association with the above enumerated symptoms.

Pancreatic Calculi

Pancreatic calculi may be diagnosed when the stone attempts to pass through the duct, thereby causing colicky pain. Pancreatic colic is somewhat similar to gallstone colic except that the pain radiates to the left epigastrium and the left shoulder. Jaundice occurs infrequently, during the height of the pain; hiccoughs, vomiting, cold sweats and collapse are of frequent occurrence; free fat in the stool and glycosuria when present are an aid to the diagnosis of pancreatic calculi.

Chronic Pancreatitis: Either acute or subacute pancreatitis may become chronic. The pain may be mild or severe; the paroxysms short or prolonged, often resembling biliary colic, and differentiated from it by the seat of pain which is generally epigastric with a tendency to radiate toward the left side; also jaundice, weakness, emaciation, frequent diarrhea—the stool containing large quantities of fat—with the presence of a tender, resisting mass in the upper midabdomen, indicate pancreatic disease. *Löwy's sign* is usually positive (Two drops of 1 to 1000 epinephrine solution instilled in the eye causes dilatation of the pupil over an extended period.)



Fig 8—Polycystic pancreas

Tumors of the Pancreas

Carcinoma: This usually occurs in people past 40 years of age (the carcinomatous age). The diastase index is above 100

Symptomatology and Diagnosis: The diagnosis of carcinoma of the pancreas alone is not easily made, but when associated with carcinoma of the stomach and gallbladder, it may be suspected by the presence of stubborn dyspepsia, progressive loss of weight, anemia and colicky epigastric pain. The pain occurs most frequently during the night and is accompanied by collapse, vomiting and diarrhea. The stool contains large quantities of fat, often blood and undigested meat fibers. If the carcinoma affects the main bile duct, jaundice will manifest itself. Pressure upon the portal vein by the tumor will cause ascites. Deep-seated tenderness with the sensation of an indefinite mass to the palpating hand, and the presence of the above enumerated symptoms, plus constipation, are highly suggestive of carcinoma of the pancreas. *Painless progressive jaundice,* not preceded by colic and *associated with enlargement of the liver* and distention of *the gallbladder,* is a frequent symptom of *carcinoma of the head of the pancreas.*

Usually when the *head of the pancreas* is the seat of malignancy there is painless jaundice; when the *body of the pancreas* is affected there is a great deal of digestive disturbance, and when the *tail of the pancreas* is invaded there are signs of diabetes mellitus. An adenoma invading the islands of Langerhans may cause severe hypoglycemia.

Tumors Other Than Carcinoma: These may cause pancreatic disturbances, the presence of which may be inferred by chronic indigestion, slight jaundice, colicky pain and a resistant tender mass in the midabdomen above the umbilicus accompanied by glycosuria

Cysts: These may be *single* or multiple, large or small.

or other pathology in that region. Then the finger is inserted just as high as it will reach and the patient is asked to bear down This procedure permits the exploration of a portion of the rectum otherwise not palpable. The rectum is thus explored in order to obtain an idea as to the presence of pathologic changes in the lower bowel and of its contents, *e g.*, impacted feces, malignant and benign growth, and foreign bodies. The amount of distention, the condition of the sphincter ani and of the adjacent structures, *i. e.*, the bladder, prostate and seminal vesicles in the male, and the uterus and other pelvic organs in the female, can thus also be learned. In a virgin a careful rectal examination will usually obviate the necessity of a vaginal examination.

III. Instrumental Examination: This is done with a rectal speculum, an anoscope or proctoscope for low examination and the sigmoidoscope for examinations as high as the rectosigmoid junction A speculum is inserted, whereby the condition of the rectal mucous membrane and the contents of the lower gut can be inspected. Internal hemorrhoids, ulcers, the condition of the crypts of Morgagni and all other visible conditions can be inspected and if deemed advisable, treated.

Diseases of the Intestines

The intestine may become affected because of Displacement from its normal position; inflammation of its mucosa; dilatation; tumors, and obstruction.

Displacement From Normal Position

The intestinal tract as a whole or any of its parts may become displaced.

(*a*) **The Displacement of the Intestines as a Whole (Enteroptosis):** The descent of the intestines is usually associated with gastroptosis and generally visceroptosis (SEE: *Glénard's disease*, p. 649, and *Gastrectasis*, p. 647).

(*b*) **Cecum:** The cecum may be displaced downward (ptosis) or it may be displaced upward, in rare instances, as high as the splenic flexure.

Symptomatology and Diagnosis: Such symptoms as constipation, colicky pains simulating appendicitis, vague digestive disturbances with an indefinite palpable mass in the right lower abdomen, and the absence of definite tenderness over the appendix, normal blood count and gastric secretion speak for disturbance in the cecum; however, an x-ray study should be made to confirm the diagnosis.

(*c*) **Redundant Colon:** This is a condition in which the colon becomes displaced; its lumen usually enlarges and is often the seat of stasis, causing putrefaction.

Symptomatology and Diagnosis: When the ascending colon is affected, cramps, constipation, indigestion and a sense of fullness in the right abdomen, which at times may simulate chronic appendicitis or nephrolithiasis, are symptomatic of this condition.

(*d*) **The Hepatic Flexure:** Because of adhesions from the gallbladder, duodenum, pancreas, or because of displacement by a large liver or kidney, this may become displaced and the seat of retention.

Symptomatology and Diagnosis: Indigestion, pain referable to the right upper abdomen, constipation and a sense of either fullness or uneasiness in the upper abdomen, with palpable rigidity of the upper rectus abdominis, are characteristic features. This condition may be mistaken for cholecystitis, duodenal

CHAPTER XXIII

Examination and Diseases of the Intestines

Physical Examination of the Intestines

By *inspection* may be determined the degree of distention or collapse of the various portions of the intestines; by *palpation* is ascertained the presence or absence of tumor masses, the amount of resistance and the presence or absence of tenderness overlying the various portions of the gut. Tenderness elicited over the abdomen when investigating the intestines is due, in most instances, to associated peritonitis, which in turn causes rigidity of the abdominal muscles. The sensation of a "doughy" mass is significant of accumulation of fecal matter in the intestines. Spastic colitis may be suspected when a sausagelike colon is palpated. An accumulation of gas is noted by the sense of elasticity it imparts to the palpating hand, also by the gurgling which it causes (SEE: *Palpation of Abdomen,* p. 586).

Percussion may determine the state of the bowel, whether it is empty or filled with gas, or solids; intestines filled with solid material or when empty will give rise to a dull note, while over a bowel distended with gas a loud closed tympanitic note will be elicited.

By *auscultation* is determined the presence of peristaltic movements; the absence of peristaltic movements may denote paralysis of the bowel, or obstruction due to any cause.

Physical Examination of the Rectum

The rectum is examined in three successive steps: (I) Inspection of the anal ring and perineum; (II) digital examination; (III) instrumental examination.

I. Inspection of the Anal Ring: By this method one can determine the presence of external hemorrhoids, fissures, malignant tumors, condylomata, ulcerations, pemphigus vegetans, pruriginous eruptions, prolapses, fistula in ano and ischiorectal abscess.

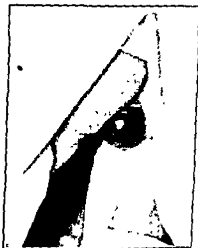


Fig. 1—Prolapsed rectum

Inspection is best accomplished by having the patient in the knee-chest position or lying on one side, the upper thigh being flexed. The part under examination should face a good light

II. Digital Examination: The patient should be in the knee-chest position or lying on one side, the upper leg and thigh flexed so as to expose as much as possible of the part under examination (the dorsal decubitus with thighs flexed is preferred by some examiners). The gloved, lubricated index finger is slowly passed upward through the anus into the rectum. First, the tip of the finger slowly sweeps the inner margin of the anus so as to palpate for internal hemorrhoids

or other pathology in that region. Then the finger is inserted just as high as it will reach and the patient is asked to bear down This procedure permits the exploration of a portion of the rectum otherwise not palpable. The rectum is thus explored in order to obtain an idea as to the presence of pathologic changes in the lower bowel and of its contents, *e.g.*, impacted feces, malignant and benign growth, and foreign bodies. The amount of distention, the condition of the sphincter ani and of the adjacent structures, *i.e.*, the bladder, prostate and seminal vesicles in the male, and the uterus and other pelvic organs in the female, can thus also be learned. In a virgin a careful rectal examination will usually obviate the necessity of a vaginal examination.

III. **Instrumental Examination:** This is done with a rectal speculum, an anoscope or proctoscope for low examination and the sigmoidoscope for examinations as high as the rectosigmoid junction A speculum is inserted, whereby the condition of the rectal mucous membrane and the contents of the lower gut can be inspected. Internal hemorrhoids, ulcers, the condition of the crypts of Morgagni and all other visible conditions can be inspected and if deemed advisable, treated

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The intestinal tract as a whole or any of its parts may become displaced.

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Symptomatology and Diagnosis: Such symptoms as constipation, colicky pains simulating appendicitis, vague digestive disturbances with an indefinite palpable mass in the right lower abdomen, and the absence of definite tenderness over the appendix, normal blood count and gastric secretion speak for disturbance in the cecum; however, an x-ray study should be made to confirm the diagnosis.

(*c*) **Redundant Colon:** This is a condition in which the colon becomes displaced; its lumen usually enlarges and is often the seat of stasis, causing putrefaction.

Symptomatology and Diagnosis: When the ascending colon is affected, cramps, constipation, indigestion and a sense of fullness in the right abdomen, which at times may simulate chronic appendicitis or nephrolithiasis, are symptomatic of this condition.

(*d*) **The Hepatic Flexure:** Because of adhesions from the gallbladder, duodenum, pancreas, or because of displacement by a large liver or kidney, this may become displaced and the seat of retention

Symptomatology and Diagnosis: Indigestion, pain referable to the right upper abdomen, constipation and a sense of either fullness or uneasiness in the upper abdomen, with palpable rigidity of the upper rectus abdominis, are characteristic features. This condition may be mistaken for cholecystitis, duodenal

ulcer, hydronephrosis or some inflammatory condition of the liver, but may be differentiated from them by the absence of colicky pains and the increased peristalsis of the transverse and descending colon and the absence of other phenomena associated with acute disease. With the aid of an x-ray examination, a diagnosis of distortion of the hepatic flexure may be made.

(*e*) **The Transverse Colon:** This is often displaced downward and in extreme cases may descend to the level of the pelvis. It usually causes stasis of the intestines, putrefaction and constipation, *which often accounts for indigestion and* nervous phenomena. The diagnosis of this condition in a patient who has vague digestive disturbances may be made by an x-ray study of the colon.

(*f*) **Splenic Flexure:** Displacement of the splenic flexure may be accompanied by dilatation or constriction and may be caused by the pressure of a large spleen or a large kidney upon this portion of the bowel, or by adhesions in other parts of the large bowel, pulling and distorting the splenic flexure.

Symptomatology and Diagnosis: The symptoms usually encountered are digestive disturbances, eructation of gas, a sense of fullness in the left upper abdomen, referred to the diaphragm and often to the precordium, associated with constipation.

Palpation may reveal slight rigidity of the left rectus abdominis, and distinct tenderness on pressure.

Percussion will yield circumscribed tympany adjacent to the stomach; this depends largely upon the amount of dilatation and degree of displacement. It should be differentiated from hypernephroma, hourglass stomach, evisceration or ventral hernia. A correct diagnosis can only be made by an x-ray examination of the colon

(*g*) **Sigmoid:** The sigmoid may become dilated because of chronic constipation or intestinal stasis; it may be displaced by tumors or adhesions, or it may become sausage-shaped.

Symptomatology and Diagnosis: The commonest symptoms are constipation, fecal impaction, vague pains in the left lower abdomen, often associated with tenesmus. When the colon is filled, a soft sausagelike mass may be palpable, and a rectal examination will reveal impacted feces. Dilatation of the sigmoid is usually free from pain or tenderness X-ray examination of the colon may reveal this condition. It is well to bear in mind that the condition of the large intestine may only be determined by physical examination when the abdominal muscles are thin and the abdomen is not distended

(*h*) **Duodenum:** The duodenum may be displaced by adhesions, large gallbladder, large kidney, hypernephroma, cyst and large liver, or any inflammatory condition in the right upper quadrant.

Diagnosis by physical examination is not possible. The symptoms may be referable either to the gallbladder or the stomach, and are sometimes associated with jaundice. The diagnosis of this condition may be made by an x-ray study of the gastrointestinal tract.

(*i*) ***Displacement of the Jejunum* and Ileum:** This cannot be diagnosed by a physical examination; there are many conditions that may cause displacement of the small intestines, *i. e.*, matting of the intestines caused by disease of the omentum, tuberculosis, peritonitis, general carcinomatosis, or tumors. The symptoms of displacement of

the small intestines are not definite because the symptoms of the underlying conditions are the predominating features

Inflammation of the Intestinal Mucosa

Acute Catarrhal Enteritis: This may be caused by indiscretion in diet, such as decomposed food and irritating poisons Hot weather (particularly for children), and exhaustion are predisposing factors It may also occur secondary to infectious diseases, portal engorgement (as a result of diseases of the heart and liver) and by extension from abnormal condition in the abdomen. Bacterial invasion and food allergy are also frequent causes of this condition

Symptomatology and Diagnosis: Diarrhea is the commonest symptom and it may be associated with cramps, a mild gaseous distention of the abdomen, borborygmi and vomiting. In some instances only a portion of the gastrointestinal mucosa may be affected, *i e.:*

Duodenum (duodenitis): When this alone is affected, the most prominent symptoms are pain and tenderness, with some discomfort localized over the upper right abdomen and associated with constipation. As a general rule, this condition is also associated with gastritis, producing the following symptoms: Anorexia; nausea; bilious vomiting; vague gastric pain, and jaundice.

Jejunum and Ileum: The existence of inflammation of the small intestine alone may be inferred by the absence of diarrhea and the presence of colicky pains, borborygmi, moderate distention of the abdomen and tenderness over the mid-abdomen, which is relieved by pressure and accentuated at the moment pressure is removed The stools are not formed, are semisolid or flocculent, and contain undigested food, small quantities of mucus and unchanged bile.

Colon: Inflammation of the large intestine is characterized by pain, profuse diarrhea with tenderness along the colon. The stool is thin, watery, containing small masses of fecal matter and large quantities of mucus (SEE: *Colitis,* p 663).

Rectum (proctitis): Inflammation of the rectum may be inferred by the presence of tenesmus, large quantities of mucus, pus and sometimes blood, either in the feces or independent of it.

Chronic Catarrhal Enteritis: This may result from repeated attacks of acute enteritis, passive congestion of the bowel due to cardiac decompensation, portal congestion and bacterial invasions.

Symptoms: These consist of chronic diarrhea which may alternate with constipation, colicky pains and abdominal tenderness The stool may contain undigested food, mucus and shreds of the intestinal mucosa The quantity may be exceedingly small or very large, and may be associated with tenesmus. Prolonged cases may develop emaciation, anemia and nervous symptoms

Infantile Diarrhea: This usually occurs in the hot months of the year in children between one and two years of age, especially in those who are artificially fed.

1. ***Acute Fermentative Diarrhea:*** This is characterized by fever, offensive diarrhea; the stool is greenish and contains undigested milk and other food with small quantities of mucus. The number of stools may vary from 3 to 20 or more daily. This condition usually occurs after taking spoiled milk or other indigestible foods, unripe or overripe fruits, or because of other dietary indis-

cretion, such as eating too much or too often.

2. *Cholera Infantum* (summer complaint): This usually occurs in children between the ages of ½ to 2 years during the hot weather (second summer). It is ushered in abruptly with persistent vomiting and severe copious diarrhea of from 8 to 30 or more stools daily. The stool is at first offensive and dark in color; it later becomes watery, odorless and alkaline, and is propelled with force.

Extreme weakness, rapid emaciation and high fever with prostration are among the characteristic symptoms.

Acute Enterocolitis: This is characterized by a follicular ulceration of the ileum, the colon and often of the entire intestinal tract. This condition usually occurs during the summer and may follow infectious diseases or other forms of diarrhea. It is ushered in with a rising temperature and diarrhea, 15 to 30 stools per day, passed without pain, seldom offensive, usually blood streaked and containing much mucus, bacillus dysenteriae, streptococci and other bacilli.

Symptomatology: This consists of abdominal distention and pain with slight rigidity and tenderness along the colon.

Celiac Disease (Gee): This is usually found in children between the ages of one to five. It is characterized by large, light-colored, gruellike, frothy, fermenting and offensive stools (diarrhea alba or diarrhea chylosa). It is not associated with fever, but anemia and wasting usually result. The abdomen has a peculiar doughy and inelastic feel, resembling tubercular peritonitis. It is possibly due to vitamin D deficiency.

Sprue or Psilosis: This is a tropical disease due to vitamin B deficiency and to the invasion by a variety of mold (monilia). It is characterized by diarrhea consisting of large, light-colored, acid stools containing large quantities of fat, and is not associated with pain or tenesmus. The tongue may be inflamed, eroded and cracked. Anemia, resembling the pernicious type, is usually present.

Diphtheroid or Croupous Enteritis: A croupous or diphtheritic inflammation of the mucosa of the entire intestinal tract may occur as the result of ingestion of poisons such as mercury, arsenic, or lead, or it may be secondary to infectious diseases such as pneumonia, septicemia, or typhoid fever, and it may occur as a terminal process in chronic affections of the kidney, liver and in cancer. It is characterized by diarrhea, abdominal pain, bloodstained mucous stool, which may contain shreds of mucous membrane; defecation is occasionally associated with tenesmus.

Phlegmonous Enteritis: This is a suppurative inflammation of the mucous membrane of the intestine associated with intestinal obstruction, strangulated hernia and intussusception It is a rare condition, affecting the duodenum more frequently than other parts of the intestinal tract. The diagnosis may be suspected when diarrhea, pus, shreds of the mucosa occur in conjunction with intestinal obstruction.

Ulceration of the Intestines: This may be due to tuberculosis, syphilis, typhoid fever, parasites and foreign bodies in the intestines. Ulcerations may also occur idiopathically or they may be due to some deficiency factor or to food allergy.

Symptomatology: The diagnostic features are those of ulceration of the intestines, irrespective of its etiology. It is characterized by diarrhea, pus and blood in the stool, sometimes actual hemorrhage may occur if the ulcer has

perforated a blood vessel. Pain and tenderness are found over the area most affected Deep ulcerations may lead to perforation of the bowel, which is diagnosed by collapse, rapid pulse, pain and sudden abdominal distention

Regional Ileitis (Crohn's disease): This is a disease of a segment of the ileum in which the mucous membrane becomes inflamed and ulcerates. The affected portion of the bowel becomes thick, edematous and rigid and the lumen becomes progressively narrowed The adjacent mesentery becomes thickened and the lymph glands enlarge. This condition is found most often in the terminal ileum, but may spread to the cecum and other portions of the bowel, or it may cause adhesions to and may ulcerate into the adjacent bowel.

Symptoms: These are of chronic progressive obstruction such as frequent colicky pain of increasing severity and of greater frequency. The pain is usually centered around the umbilicus and the right lower quadrant of the abdomen, associated with general distention Diarrhea alternates with constipation and there is occasional vomiting The stool contains occult blood and when loose it contains mucous shreds

Physical Examination: In moderately advanced cases this reveals the patient to be pale and to have evidence of loss of weight, the abdomen is distended and there is tenderness and a sausagelike rigidity or mass in the right iliac fossa The temperature is somewhat elevated A blood examination will reveal in most cases a hyperchromic macrocytic anemia with a slight polymorphonuclear leukocytosis The x-ray examination is a valuable diagnostic aid when carefully done. This condition is to be differentiated from subacute appendicitis, ileocecal tuberculosis and carcinoma of the ileum.

Appendicitis

Appendicitis is an acute inflammation of the vermiform appendix. This condition may be caused by the lodging of a foreign body in its lumen, by bacterial invasion and inflammation of its mucosa from any cause. Parasites and carcinoma may also be among the causative features

Three stages of appendicitis are recognized:

1. Acute catarrhal appendicitis.
2. Chronic catarrhal appendicitis.
3. Acute purulent appendicitis.

Symptomatology and Diagnosis: 1. *Acute catarrhal appendicitis* presents a slight rise in temperature, pain over the right lower abdomen at McBurney's point. It should be borne in mind that the appendix may be displaced upward toward the gallbladder; it may be retrocecal or it may be pulled over toward the left, or it may be found in the left iliac region (situs inversus); these abnormal positions should be borne in mind when the site of abdominal pain is considered in the diagnosis of appendicitis Tenderness and rigidity of the lower part of the right rectus abdominis is, however, a most frequent occurrence Vomiting does not usually occur at this stage.

2. *Chronic catarrhal appendicitis* is characterized by vague abdominal pain, digestive disturbances, and some tenderness on deep pressure over the site of the appendix.

3. *Acute purulent appendicitis* is ushered in abruptly with fever, vomiting, severe agonizing pain over the appendiceal region, associated with tenderness

Differential Diagnosis of Extrauterine Pregnancy, Salpingitis and Appendicitis

Symptoms	Extrauterine Pregnancy	Salpingitis	Appendicitis
Pain	Comes on generally after exertion, and is sudden in onset The pain is most intense and is localized in the lower abdomen In some cases a pain is also felt in the shoulder of the same side	Pain may be gradual in onset, though in some cases it is very acute. Begins in the lower part of abdomen. In acute cases the pain is sudden in onset and is localized in the tubal areas. In generalized peritonitis pain is absent	Generally sudden in onset. At first is in the midline. Later it passes over to the right iliac fossa
Vomiting	Frequent and synchronous with the pain	Vomiting is a late symptom	Vomiting is an early symptom
Pulse	At first, because of shock, may not be greatly increased in rapidity. After the primary shock, the rapidity is not very great until the amount of blood lost becomes excessive	Generally rapid in acute lesions. In chronic lesions generally no change	Generally very rapid in acute cases
Tumor.	Very sensitive and tender and lies to one side of the uterus Is constantly increasing in size After rupture, when a hematocele has formed, the tumor mass of the uterus rapidly increases in size, and is soft and boggy	Painful swelling to one side of the uterus Generally the uterus is fixed and is not freely movable. Tumor is often bilateral.	Tumor in acute appendicitis can rarely be defined because of the excessive tenderness and rigidity of the abdominal muscles Percussion sometimes elictis tenderness when palpation fails to do so If an abscess has formed, it can be felt by vaginal examination.
History.	Of pregnancy, with enlargement of the uterus which is not in proportion to the stage of the pregnancy	History of recent childbirth or of a vaginal infection. Often no accountable cause is present	History of previous attack may be present
Temperature	No elevation. Generally normal.	Rise of temperature	Generally sudden, progressive rise
Uterus	Enlarged	Not enlarged	Not enlarged.
Blood	Hemoglobin low and decreasing. Red and white cells both reduced.	Hemoglobin high, whites increased, reds normal	Leukocytosis always present. Hemoglobin and red cells normal
Abdomen	Fluid, if the hemorrhage has been very great, may be elicited on palpation and percussion Puncture of the posterior vaginal vault with an aspirating needle frequently will reveal condition. A mass is present in pelvis Rigidity of abdominal muscles may be present. No change in intestinal peristalsis.	N [illegible] change in intestinal peristalsis	[illegible] present Mass [illegible] region [illegible] in ab- [illegible] No [illegible] talsis Localized rigidity over lower segment of rectus.

Differential Diagnosis of Perirenal Abscess, Osteomyelitis and Suppurative Appendicitis

Symptoms	Perirenal Abscess	Osteomyelitis (vertebra)	Appendicitis (abscess formation)
Pain.	Rather severe Tenderness is most marked on pressure made in the subcostal angle Tenderness also is felt on pressure made through the anterior abdominal wall The pain is eased by flexion of the vertebra The pain radiates down in the direction of the ureter.	Not very severe. Tenderness is most marked on pressure made over the affected vertebra. Very little tenderness is felt on pressure through the anterior abdominal wall. Pain may radiate down to the hip, when the abscess reaches the psoas muscle it runs along this muscle to the hip.	History of a very severe pain Generally at the time the patient comes under observation the pain may be so severe and resembles perirenal abscess Pain may be produced by the taking of food For further pain, see Appendicitis, page 661.
Vertebrae.	Fixity of vertebrae absent	Fixity of vertebrae. In tuberculous disease of the vertebra kyphosis is present as a late symptom	No rigidity of the vertebra.
Time of development.	May be fairly rapid	Slow.	May be slow or rapid Follows an acute attack of appendicitis.
Urine	Pus; blood generally found if examinations are persistently and carefully made	Pus and blood in urine are absent.	No pus, nor blood, etc , present
Nausea and vomiting.	Common	Unusual	Common
Tumor.	P [illegible] can be felt sometimes through the anterior abdominal wall	[illegible] pearance may closely resemble the tumor mass of a perinephritic abscess	Tumor mass is lower down than in perinephritis Is best felt from in front Is rather sharply circumscribed

to pressure and rigidity over the right lower abdomen. Leukocytosis is always present.

Appendicitis should be differentiated in women from extrauterine pregnancy and salpingitis, also from perirenal abscess and osteomyelitis vertebrae.

Colitis

Colitis is an inflammation of the colon which may be regional or diffuse, specific or nonspecific. Disease of the colon occurs most often because of a primary injury to its wall. The injured portion, because of lack of resistance, may fall prey to a secondary invader such as one of several organisms found in the feces or in the circulating blood. Primary injury to the colon may be brought about by a number of conditions: 1 Vascular, *i. e*, emboli, thrombi or other conditions interfering with proper nutrition of a large or a small portion of the colon; 2 Lymphatic, *i e*, disturbance in the lymphatic circulation of the bowel which may greatly interfere with the surface

tension of the colonic mucosa and its function; 3. Nervous, *i e.*, *interference* with the autonomic balance by causing greater spasticity as in vagatonia or greater dilatation as in sympatheticotonia and thus also interfering with its vascular tone and possibly with its protective secretion; 4. Irritating substances in the stool, either mechanical or chemical; 5. Neoplasm, benign or malignant; 6. Syphilis, 7. Primary bacterial or parasitic infections, *i. e., the endamoeba, tuberculosis*; 8. Vitamin deficiency, *i. e., sprue, pellagra, etc.*

Symptomatology: Regardless of the cause, colonic irritation is manifested clinically by a change in the number and consistency of the daily evacuations and in the production of an excessive secretion and the expulsion of mucus, of mucoid substances and, occasionally, of blood. Abdominal pain of various types, degrees, and in various locations may or may not be present. Pathologically the changes vary with the severity of the irritation; the various portions of the colon may be spastic or relaxed, contracted or dilated, and its mucosa may be inflamed, ulcerated or may appear normal.

While these *general symptoms* are found in all types of colitis, there are also specific, local and constitutional manifestations that are characteristic of the various types or stages of the disease. Because of the varied etiology, the divergent pathology and the multiformity of the clinical manifestations, colitis may be classified as acute and chronic, and as the specific, *i. e.*, of known etiology, and the nonspecific, *i. e.*, of unknown or doubtful etiology.

Among the specific types of colitis may be mentioned those that have a definite etiology irrespective of the type of lesion, *i e.*, carcinomatous, tuberculous, syphilitic, bacillary, amebic and other tropical types, as well as those resulting from corrosive poisoning and mechanical injury caused by foreign bodies.

The nonspecific or so-called idiopathic colitis may be divided into four groups: 1. Spastic or functional colitis or irritable colon; 2 Colosis or mucous colitis; 3 Idiopathic, ulcerative or inflammatory colitis, and 4. Allergic colitis. Whether *these are four distinct entities or progressive stages of the same disease is still open to question*

Spastic or Functional Colitis or Irritable Colon: This condition gives rise to a train of local and systemic manifestations and to reflex phenomena which may be referred to *distant organs*, or to the individual as a whole These symptoms may be vague or definite, specific or contradictory. The physical signs are also inconstant and proctoscopic examination usually *reveals nothing* pathologic X-ray examinations, however, are of great diagnostic value.

Symptomatology: The patient is, as a rule, extremely sensitive, irritable, easily annoyed and fatigued. The chief complaints are those of indigestion, passing of gas at both ends, pyrosis, borborygmus and constipation, or constipation alternating with diarrhea. Mild purgation may set up a severe diarrhea at one time, while at another time a drastic purgative will cause only a scant bowel movement. The reaction to an enema is also variable; some patients are capable of tolerating only a small quantity of fluid, while others may hold three or four quarts with comfort. A certain number of them are distressed or become faint when an enema is passed.

Pain is variable; it may be generalized over the entire abdomen as a sense

of fullness or discomfort, or it may be acute in the right or left upper or right or left lower quadrants of the abdomen. Because of the distribution of the pain, the signs of indigestion, and the general nervousness of the patient, this condition is frequently mistaken for cholecystitis, pancreatitis, renal calculi and appendicitis. Other manifestations such as insomnia, headache, tiredness and particularly cardiac palpitation, heart sensitiveness and other neurogenic expressions are common in this condition

Physical Examination: A physical examination reveals general or shifting areas of abdominal tenderness, the area of tenderness often depending upon the degree of distention of a circumscribed portion of the bowel. There is usually no muscle rigidity nor are there areas of skin hypersensitivity. The bowel content may be loose and of offensive odor, or it may be of various degrees of hardness, sometimes even stony hard and black It may be passed in scybalous masses, or it may be cylindrical, varying in the size of its circumference and contour.

Proctoscopic examination is usually negative.

X-ray examination with an opaque enema, or an opaque enema followed by an air enema may reveal numerous colonic defects in contour but not in the mucosa. The entire colon may show spasticity with marked contractions of its haustrae, or the haustrae may be entirely absent so that the sigmoid presents the so-called plumber's pipe appearance. The colon may be redundant or hugely dilated throughout its course or it may be dilated in some parts and contracted in others There may also be displacement of the transverse colon and sigmoid. The point worth noting here is that repeated roentgenographic examinations of the colon may show a divergent picture at each examination.

Etiology: There are probably several factors operative in the production of functional or irritable colon The more obvious ones are: (*a*) A familial tendency or heredity; (*b*) psychic disturbances; (*c*) autonomic imbalance; (*d*) constitutional anomalies, and (*e*) chronic cardiac and renal disease.

Colosis or Mucous Colitis: The term colosis, I believe, is more applicable because of the absence of any definite evidence of inflammation of the mucosa, musculature or any other structure of the colon. The change of the ending "itis" to "osis" is here preferred because it indicates cloudy swelling rather than inflammation and is similar to the nomenclature adopted in the differentiation between nephritis and nephrosis or carditis and cardosis

Colosis occurs more frequently in women than in men, usually between the ages of 18 and 30 years. It is generally associated with other constitutional derangements, often of an endocrine basis Sufferers from this type of colon dysfunction frequently show evidence of hypopituitarism which in the female manifests itself by dysmenorrhea or periods of amenorrhea, or other functional ovarian disturbance The basal metabolic rate is, as a rule, subnormal, indicating also some hypothyroidism The cholesterol content of the blood is increased and not infrequently one finds an increased serum globulin Functional neurosis is definitely associated with this condition. Whether the neurosis is the primary condition responsible for the bowel dysfunction or the colon disturb-

ance causes the individual to become neurosensitive is an undecided question

Symptoms: The symptoms are of two types; one is constitutional, and the other directly referable to the gastrointestinal tract The *constitutional symptoms* are nervousness, excitability, restlessness, fatigue, ready exhaustibility, occasionally associated with insomnia, disturbing dreams, paresthesia of the extremities with occasional involuntary movements. There may also be palpitation or other cardiac arrhythmias and headaches. The individual is as a rule not thin. Occasionally there are complaints of having lost a considerable amount of weight The patient appears pale, but the blood picture only rarely discloses any anemia The *gastrointestinal symptoms* are anorexia, alternating occasionally with excessive appetite. There is generally a sense of epigastric or abdominal fullness, with mild colicky pains or some discomfort in the lower abdomen. The pain may at times be quite severe and localized so that a diagnosis of appendicitis, gallstones, and, in women, pelvic inflammatory disease is made. The patient may be entirely constipated. Occasionally large quantities of mucus are passed without any feces, but at other times there may be just a thin serous discharge which causes burning of the rectum. Flatulence as well as tenesmus are frequent symptoms Blood in the bowel movement is rare and found only on occasion when large shreds of mucus have been forcibly torn away because of drastic purgation.

Physical Examination: The abdomen may be either greatly distended or definitely scaphoid. In the constipated cases where the colon is overfilled with gas and fecal matter, the abdomen is distended, while those suffering from diarrhea may or may not have a scaphoid abdomen. However, in all cases of colosis, whether they are constipated or have diarrhea, the abdomen is enlarged By enlargement I do not mean distention. The enlargement may be noted by measuring the distance between the anterior superior spine of the ilia and the lower costal angles on each side where a definite increase in measurements above the normal will be readily detected. This is caused not so much by the abdominal distention as by the relaxation of the spinal muscles which are responsible for abdominal contour. *Palpation* of the abdomen may elicit tender areas along the ascending and descending colon and occasionally sausagelike masses may be detected over these areas The transverse colon is seldom palpable. Reflex contraction of the anus often adds greatly to the individual's suffering not only because of tenesmus but because of its interference with the passage of the bowel content. On sigmoidoscopic examination the mucous membrane appears edematous, pale, and distended, the surface presenting a pitted appearance where the shreds of mucus were dislodged, but no actual ulcerations or bleeding points are visible.

Diagnosis: The diagnosis of colosis is based upon the history of gastric disturbance with manifestations of lower abdominal disturbance, the passage of large quantities of mucus by bowel, the presence of diarrhea or of constipation, the absence of blood and specific organisms in the fecal matter, the presence of tender areas along the colon and the characteristic proctoscopic findings Constitutional symptoms such as fever, anemia and prostration are usually absent. This condition should be differentiated from chronic appendicitis, di-

verticulosis, spastic colon, ulcerative colitis, enterocolitis and the various types of specific colitis.

Idiopathic Ulcerative Colitis—Colitis Gravis—Hemorrhagic Colitis or Idiopathic Organic Colitis: Ulcerative colitis may be defined as a chronic suppurative disease of the colon, characterized clinically by tenesmus, unformed stools containing mucus, pus and blood. Sigmoidoscopically it is evidenced by the presence of superficial and deep ulcers in the colonic mucosa which are partially covered with mucus and pus and surrounded by inflammatory areas

Pathology: The lowermost portion of the colon is usually affected, presenting sigmoidoscopically a variety of lesions. Inflammatory changes, areas of edema, minute abscesses and ulcerations may be seen at various times and at various points. The size of the lesions are as variable as are their number.

Symptomatology: The symptoms and physical signs depend somewhat upon the severity of the disease. In all cases there is some abdominal pain, either severe or mild; the bowel movements are thin, containing mucus, pus and blood, the number of stools are variable, ranging from two or three to 20 or more per day. There is usually a rise in temperature; secondary anemia develops quite early and is often marked. There is a gradual or rapid loss of weight and profound nervous irritability. The abdomen is tender to touch and imparts a sense of resistance, but there are seldom, if ever, any areas of painful rigidity. Exacerbations and remissions may occur spontaneously

Diagnosis and Differential Diagnosis: The diagnosis is based upon the rather gradual onset and the progression of symptoms, the proctoscopic findings, the x-ray findings and the bacteriologic examination. Before a diagnosis of ulcerative colitis is made, it is necessary to exclude the many conditions simulating it. Among the most important to be borne in mind are the various types of bacillary dysentery. amebic dysentery, carcinoma of the colon, tuberculous enterocolitis, diverticulitis, thyroid crisis, and allergic colitis

Allergic Colitis: It is often noted that allergic reactions manifest themselves in the colon as well as in other parts of the body. Occasionally, the entire gastrointestinal tract plus the colon may be equally affected. Persons who are subject to urticaria, to migraine and to other allergic phenomena frequently develop profuse diarrhea which is occasionally mixed with bloody discharge. Proctoscopic examination during that time will reveal circumscribed areas of congestion in the colon. These can be made to disappear temporarily by the hypodermic injection of adrenalin chloride solution or by the local application of epinephrine or ephedrine solutions When such patients are tested for their allergic sensitivities, the cause of the diarrhea may or may not be found.

Dilatation

Dilatation of the colon (megalocolon) may be acute or chronic Acute dilatation of the colon may result from acute intestinal obstruction, acute gastroenteritis, and paralysis of the bowel, it may occasionally occur in conjunction with distention of the entire intestinal tract as seen in typhoid fever and pneumonia Chronic intestinal dilatation may be congenital as in Hirschsprung's disease or it may be acquired because of chronic constipation, slowly growing colonic tu-

mor or other conditions causing partial obstruction with paralysis of the gut.

Hirschsprung's Disease: This is an idiopathic dilation of the colon appearing during early childhood and may be *carried over into adulthood; it is commoner among boys.* The usual site is the



Fig. 2—Hirschsprung's disease.

sigmoid flexure, which may be enormously distended; occasionally the entire colon may be affected. There is usually an associated achalasia of the rectum with hypertrophy of the muscular coat of the pelvic colon and rectum. This condition may be brought about by some disturbance in the autonomic innervation of the sigmoid or by inflammation of Auerbach's plexus (Munro Cameron).

Symptomatology: The abdomen is greatly distended, there is obstinate constipation; the intervals between bowel movements may be several days, a week or longer. Often diarrhea alternates with constipation and there may be signs of colitis.

Diverticulitis: Diverticula may be congenital or acquired They are pouch-like dilatations of the colon and may be single or multiple. *Meckel's diverticulum* is usually found some distance above the ileocecal valve and may be attached to the umbilicus This may cause entangling of the bowel and lead to intestinal obstruction. *Occasionally when* inflamed it may resemble acute appendicitis; the pain and rigidity is more marked in the umbilical region than over the right rectus. *Multiple diverticula* may be found in the colon and at times in the duodenum; the most common seat is the sigmoid. Occasionally they may become inflamed and produce symptoms of partial obstruction, *i. e*, pain, diarrhea or constipation. On palpation either a sense of resistance or a sausage-shaped mass may be felt in the left lower quadrant of the abdomen. The diagnosis of this condition may be made by an x-ray study of the colon.

Mesenteric Thrombosis or Embolism

Mesenteric Thrombosis or Embolism is characterized by acute abdominal pain, distention of the abdomen and often by shock, hematemesis and melena. It may resemble perforation of the bowel, acute pancreatitis, perforation of a gastric ulcer, acute intestinal obstruction or lead colic.

Tumors of the Bowel (Benign and Malignant)

(*a*) **Benign Tumors:** These manifest themselves by causing partial obstruction of the bowel either because of their presence within the lumen of the gut or by compression from without

Diagnosis: Benign tumor is seldom diagnosed by a physical examination unless the tumor is so large that it may be palpable. Benign tumors such as hydronephrosis, hepatic tumors, cysts and abscesses, distended gallbladder, enlarged abdominal lymph glands, enlarged omental glands, aneurysm of the abdominal aorta, psoas abscess, tuberculous

abscess of the vertebra, ovarian cyst, intestinal tumors and teratomata, also splenic enlargement, enlarged kidney, cyst of the kidney and large liver may cause partial obstruction of that part of the bowel with which it comes in contact.



Fig 3—Carcinoma of transverse colon.

(*b*) Malignant Tumors: *Carcinoma* of the colon is a fairly frequent disease and gives rise to symptoms of partial compression plus cachexia. Occasionally severe colicky pain may precede the other symptoms for some time. Malignancy of the intestine occurs most frequently at the transverse colon, descending colon, the sigmoid and rectum Characteristic signs are abdominal cramps, diarrhea, bloody stool, associated with or without tenesmus and shreds in the stool. In some instances constipation is marked. An x-ray examination will, as a rule, reveal obstruction.

Sarcoma usually affects the small intestine and originates from beneath the mucosa. The mesenteric and the retroperitoneal glands may be the seat of such infection, it is more frequently found in children and young adults (Fig. 19, p. 591)

Lobstein's cancer is a primary retroperitoneal lymphosarcoma. It usually lies deep in the abdomen in a transverse position and is fixed. There is usually severe, persistent and deep-seated pain often referred to the back. It generally affects children.

Malignancy of the retroperitoneal glands may be primary or secondary. It may cause intestinal obstruction or ascites. When the spinal nerves are affected there is severe abdominal pain resembling acute appendicitis, cholecystitis, perforated peptic ulcer, acute peritonitis, renal colic, Dietl's crisis, mesenteric embolism or thrombosis or lead colic. The retroperitoneal glands may also be the seat of tuberculosis and of Hodgkin's disease. Hypernephroma, adrenal tumors, ovarian malignancy, testicular malignancy and other malignancies may also invade these glands.

Intestinal Obstruction (Ileus)
(Acute and Chronic)

Acute Obstruction: This may be caused by· 1. Strangulation; 2 Intussusception; 3 Volvulus or torsion

1. *Strangulation* occurs as a result of a loop of intestine being caught between abdominal adhesions, adherent appendix, mesenteric or omental slits, and pedunculated tumors; or the bowel may be forced through a hernial ring.

2 *Intussusception* is an invagination of adjacent parts of the bowel where one portion of the gut is telescoped into another with subsequent constriction due to tumefaction, resulting in obstructions Invagination of the bowel usually occurs at the ileocecal valve though it may occur in the ileum or colon alone; or it may be confined to the large intestine and may be colicorectal, in which instance the colon and rectum are in-

mor or other conditions causing partial obstruction with paralysis of the gut.

Hirschsprung's Disease: This is an idiopathic dilation of the colon appearing during early childhood and may be carried over into adulthood; it is commoner among boys. The usual site is the



Fig 2—Hirschsprung's disease.

sigmoid flexure, which may be enormously distended; occasionally the entire colon may be affected. There is usually an associated achalasia of the rectum with hypertrophy of the muscular coat of the pelvic colon and rectum. This condition may be brought about by some disturbance in the autonomic innervation of the sigmoid or by inflammation of Auerbach's plexus (Munro Cameron).

Symptomatology: The abdomen is greatly distended; there is obstinate constipation; the intervals between bowel movements may be several days, a week or longer. Often diarrhea alternates with constipation and there may be signs of colitis.

Diverticulitis: Diverticula may be congenital or acquired They are pouch-like dilatations of the colon and may be single or multiple. *Meckel's diverticulum is usually found some distance above* the ileocecal valve and may be attached to the umbilicus. This may cause entangling of the bowel and lead to intestinal obstruction. Occasionally when inflamed it may resemble acute appendicitis; the pain and rigidity is more marked in the umbilical region than over the right rectus. *Multiple diverticula* may be found in the colon and at times in the duodenum; the most common seat is the sigmoid. Occasionally they may become inflamed and produce symptoms of partial obstruction, *i. e.*, pain, diarrhea or constipation. On palpation either a sense of resistance or a sausage-shaped mass may be felt in the left lower quadrant of the abdomen. The diagnosis of this condition may be made by an x-ray study of the colon.

Mesenteric Thrombosis or Embolism

Mesenteric Thrombosis or Embolism is characterized by acute abdominal pain, distention of the abdomen and often by shock, hematemesis and melena. It may resemble perforation of the bowel, acute pancreatitis, perforation of a gastric ulcer, acute intestinal obstruction or lead colic.

Tumors of the Bowel (Benign and Malignant)

(a) Benign Tumors: These manifest themselves by causing partial obstruction of the bowel either because of their presence within the lumen of the gut or by compression from without.

Diagnosis: Benign tumor is seldom diagnosed by a physical examination unless the tumor is so large that it may be palpable. Benign tumors such as hydronephrosis, hepatic tumors, cysts and abscesses, distended gallbladder, enlarged abdominal lymph glands, enlarged omental glands, aneurysm of the abdominal aorta, psoas abscess, tuberculous

Chronic Obstruction: This may be caused by a slowly growing tumor, large prostate, fecal impaction because of the gradual collection of feces in the cecum or sigmoid. Stricture due to adhesions, congenital strictures and paralysis of the bowel may cause a slowing up of peristalsis with the gradual decrease in size of the lumen of the intestine, and subsequent obstruction.

Symptomatology and Diagnosis: Prior to the final obstruction the important signs are distended abdomen with tympany, weak peristalsis, toxic symptoms such as indigestion, headache and various pains and aches throughout the body. The stool may be ribbon-shaped or it may occur in scybalous masses and may contain mucus, blood and pus. The symptoms will often depend upon the underlying cause of the chronic obstruction When complete obstruction finally occurs the signs are similar to those of acute obstruction of the bowel

Constipation

Constipation may occur as the result of improper food, because of insufficient residue, lack of fluids, bad habits such as restraining from stool, atony of the bowel, general weakness, fecal impaction, megalocolon, Hirschsprung's disease, diverticulosis, tumor of the bowel, rectal disease, intestinal obstruction, paralytic ileus and hysteria; constipation is only a symptom, its cause depends upon the underlying factors (SEE. p 92).

Symptoms Referable to the Anus and Rectum

Itching (pruritus ani): Itching of the anus is a most distressing symptom; it may be due to a variety of causes, and occasionally no cause is discoverable. The commoner causes are:

(*a*) Irritation around the anus due to low grade local infection as seen in the presence of irritating vaginal or rectal discharge, uncleanliness of the part; (*b*) skin rashes such as eczema, ringworm, herpes, neurodermatitis, nodular prurigo erythema; (*c*) parasitic infection, *i.e*, scabies, pediculosis, dermatophytosis, pinworms, roundworms; (*d*) constitutional diseases such as diabetes, jaundice, nephritis, constipation, digestive disorders, allergic manifestation, diarrhea, certain nervous affections; (*e*) local disease of the part such as proctitis, ulcer of rectum, anal fissure, hemorrhoids, fistula, papillitis and cryptitis, foreign body lodged in a crypt; (*f*) menopause and postmenopausal age—the anal itching at that age is often an extension from the vulvar or pubic itch due to endocrine disturbance, atrophy of the parts or to the degenerative process of old age; (*g*) local injury or healing of wounds either surgical or accidental which are often accompanied by intense itching.

Pain: Pain in the rectum may be constant or it may occur only during defecation or soon thereafter. Constant pain in the rectum and perineum, which is usually aggravated by defecation, may be caused by ischiorectal abscess, anal abscess, strangulated or inflammed hemorrhoids, carcinoma of the rectum, periproctitis, prostatic abscess, seminal vesiculitis, fecal impaction, acute salpingitis, tabes dorsalis causing rectal crisis, irritation of the rectum and anus by diarrhea, irritating foods, foreign bodies, fissures and rectal polyps or adenoma.

Pain during defecation is caused by fissure in ano, rectal ulcer, inflamed hemorrhoids, anal abscess, fistula in ano, stenosis or stricture of the rectum, dysentery, fecal impaction, foreign body

volved. In children intussusception of the appendix may occur, though this is not frequent.

3 *Volvulus or torsion* is a twisting of the intestine and is most frequently met with at the sigmoid flexure of the colon. A long and relaxed mesentery may predispose to this condition. As a rule a loop of the intestine is twisted upon its long axis and the portions at the end of the loop cross each other, thus causing strangulation, or one portion of the bowel may be twisted about another.

Symptomatology and Diagnosis: Acute obstruction is ushered in with severe abdominal pain, abdominal distention, absence of bowel movement, though feces in the rectum may be washed out with an enema; bloody, serous fluid, containing intestinal mucosa and mucus may constitute a stool. Vomiting, first the stomach contents, then bile, and finally the contents of the bowel (fecal or stercoraceous), and collapse may follow. Peristalsis cannot be heard beyond the seat of the obstruction

On *percussion* tympany may be elicited because of distention of the bowel above the obstruction; beyond the obstructing point dullness may be found due to empty bowel. Acute intestinal obstruction should be differentiated from acute generalized peritonitis.

Differential Table of Acute Generalized Peritonitis and Acute Intestinal Obstruction

Symptoms	Acute Generalized Peritonitis	Acute Intestinal Obstruction
History	There is a history of causal conditions or diseases (ulcer, appendicitis, pelvic infection)	There is a history of previous chronic obstruction or hernia or there may be postoperative adhesions.
Temperature	An early and considerable rise of	No early rise (except in volvulus),
Pain,		
Vomit		
Collapse	Collapse occurs late.	Earlier onset of collapse
Leukocytosis	In septic cases, leukocytosis with increase in polynuclear cells	There may be increase in number of leukocytes.
Abdominal distention	Distention of the abdomen is usually general and marked	Less marked, unless the obstruction be situated in the lower segment.
Visible peristalsis.	Visible peristaltic waves absent.	Present and pronounced when the seat of obstruction is low, and course of wave may be reversed
Tenderness.	Tenderness decided and general.	Tenderness localized and usually slight.
Effusion.	Signs of effusion appear	Less common, due to secondary peritonitis.
Auscultatory signs.	Auscultation negative	Loud gurgling and splashing sounds audible over the abdomen (colon), above the obstruction. No gurgling beyond obstruction.

swollen, bluish-red folds and the bleeding points in case of hemorrhage. In any case of bleeding from the rectum, regardless of the patient's age, a thorough rectal and sigmoidoscopic examination should be done so as to exclude carcinoma. The combined internal and external hemorrhoids often have the features of both.

Fissure in Ano: This is usually single though they may be multiple Each occurs as a small crack at the anterior or posterior commissure in a fold of the anus. Occasionally it appears as a small ulcerated area in the mucosa of the canal. It causes intense burning and lancinating pain aggravated by defecation; following defecation there is throbbing and spasm. Care must be taken during examination as the pain is too intense for instrumental or even for finger examination. Such examination should be delayed until after the acute pain has subsided.

Ulcers of the Rectum may be simple, tuberculous, syphilitic, malignant, typhoidal or dysenteric. Irrespective of its etiology a rectal ulcer usually causes tenesmus, spasm of the sphincter muscle with diarrhea and much pain The diarrhea is most pronounced on arising and may contain mucus, pus or blood Pain, whether on defecation or on motion, depends upon the site of the ulcer and its cause. The closer the ulcer is to the anus, the more severe is the pain. Digital examination, proctoscopy, biopsy, stool and blood examination may aid in the diagnosis of the underlying cause of an obscure rectal ulcer.

Fistula in Ano: This may result from a previous suppuration or from local disease; at times it is associated with pulmonary tuberculosis The opening may be internal or external or it may have several openings. It usually causes itching and irritation and some moisture around the anus. Periodically it may cause pain during defecation. This occurs only when the fistula has closed and has become distended with pus The discharge of the accumulated pus affords relief from pain. Proctoscopic examination may reveal the site of the internal opening and probing may reveal its direction.

Rectal Polypi or Adenomata: These are usually pedunculated growths, soft and dark in color. The symptoms are those of a mass in the lower bowel, such as constant desire to defecate, marked fullness or a sense of weight in the lower abdomen, pain in the perineum, lower back and down the thighs, and frequent bowel movements of small watery stool accompanied by loud flatulency and frequent micturition. When the polypi begin to degenerate, large dark offensive material is involuntarily discharged from the rectum at varying intervals Finger palpation and proctoscopic examination will usually reveal the mass.

Carcinoma of the Rectum: Carcinoma of the rectum is not confined to old people alone. Occasionally it may occur in persons in the late teens or in early adulthood. Rectal bleeding often without pain when no local cause is discoverable should be thoroughly investigated. The rectum should be examined by finger, proctoscope or sigmoidoscope. If no cause for bleeding can be found by these methods the colon or the entire gastrointestinal tract should be studied by x-rays. Other studies such as the various blood tests may in obscure cases aid in the diagnosis of melena. Carcinoma of the rectum is of two types, one an *ulcerative type* that

lodged at the anal ring, and any inflammatory condition of the rectum or its immediate vicinity.

Tenesmus: This may be defined as a painful sensation of expulsive contraction of a sphincter (bearing down). Rectal tenesmus may be caused by ulcer of the rectum, hemorrhoids, carcinoma of rectum, rectal polyps or adenoma, periproctitis, colitis, diarrhea and foreign bodies in the rectum.

Bleeding from Rectum: Bleeding from the rectum may vary in color, quantity and in its relation to the bowel content. Bright red blood usually comes from the vicinity of the rectum; dark blood usually comes from higher up in the bowel; very dark or tarry blood may come from the stomach or duodenum Small quantities of blood may come from hemorrhoids, cancer, anal fissure or ulcer Larger quantities may come from ulcerative colitis, ulceration of the bowel, carcinoma of the colon, and dysentery Large quantities of blood may come from a peptic ulcer, intestinal or gastric varices, hemophilia, purpura, aplastic anemia, nephritis, Banti's syndrome and cirrhosis of the liver In children Meckel's diverticulum is an occasional cause for melena

Rectal Discharges Other Than Blood: This may be due to some inflammatory condition of the anus, rectum or colon, or to carcinoma, abscess, syphilis, relaxed rectum or incompetent sphincter associated with colitis or other bowel suppuration of spinal cord disease.

Diseases of the Rectum and Anus

Proctitis: This is an inflammation of the rectum associated with inflammation of the lower colon. It may be of two types: (1) Hypertrophic in which there is thickening of the anal folds with hypertrophy and occasionally with local edema of the anal ring; and (2) atrophic which presents atrophy of the perianal tissue with multiple superficial fissures Both types may be due to intestinal toxemia, constipation or diarrhea. There is usually intense itching, a sensation of heat or of fullness, and tenesmus. The bowel movements are frequent, containing small masses covered with mucus, pus or blood.

Hemorrhoids (*piles*): These may be external or internal, and occasionally there is a combination of the two.

External Hemorrhoids: These are rounded or oblong varicosities of the veins surrounding the anus; they may occur singly or in number, and when distended are of a bluish cyanotic color When inflamed and strangulated by the anal sphincter they cause intense pain which becomes aggravated by defecation Healing takes place after rupture or surgical opening of the mass, which permits the extravasation of fresh and clotted blood, or by thrombosis either induced by injection of sclerosing substances or spontaneous clotting which causes organization of the hemorrhoid resulting in the formation of scars or tabs.

Internal Hemorrhoids: These are dilated varicosities or nevi originating around the internal orifice of the anus. They may cause bleeding, itching and when inflamed will cause pain on defecation When they become very large they may protrude through the sphincter ani and may become strangulated. Internal hemorrhoids are not always palpable, though usually they may be felt with the examining finger just inside the anus and may occasionally be brought out through the anus with the finger. Proctoscopic examination will reveal the

swollen, bluish-red folds and the bleeding points in case of hemorrhage. In any case of bleeding from the rectum, regardless of the patient's age, a thorough rectal and sigmoidoscopic examination should be done so as to exclude carcinoma The combined internal and external hemorrhoids often have the features of both.

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pletely covered by the peritoneum; (2) some of the viscera are partly covered by it; (3) a few structures are adjacent to the peritoneum but not covered by it; and (4) several structures are entirely devoid of peritoneum.

1. ***Abdominal Viscera Almost Completely Covered by Peritoneum:*** Stomach, spleen, liver, ascending portion of the duodenum, the jejunum, ileum, transverse colon, sigmoid flexure, upper end of rectum, uterus, ovaries, and the fallopian tubes

2. ***Abdominal Viscera Partially Covered by Peritoneum:*** The descending and transverse portions of the duodenum, cecum, ascending and descending colon, part of the vagina, and the urinary bladder (only its posterior wall is covered).

3. ***Structures Adjacent to the Peritoneum But Not Covered by It (Extraperitoneal):*** The adrenals, the kidneys, and the pancreas.

4 ***Structures Entirely Devoid of Peritoneal Covering:*** The lower part of the rectum, the base and neck of the urinary bladder, the prostate gland, and the lower part of the vagina.

The *visceral peritoneum* runs a complicated course and cannot be readily traced. During the embryonic development, the intestines undergo many changes in shape and position. Their rotation and development during the embryonic stages produce many peritoneal folds which serve as means of attachment and support for the various organs and enclose the vessels and nerves of each part as they pass to and from the posterior part of the abdomen

The Peritoneal Folds: These are of three types:

1. ***The Mesenteries:*** These are composed of two layers of serous membrane They connect the jejunum and ileum, the *mesocecum, the transverse, and the sigmoid mesocolon*, and the mesorectum to the posterior abdominal wall in front of *the vertebral column*. Between the layers of the serous membrane are carried the various branches of the mesenteric arteries and veins, the nerves, the *lacteal* vessels, and mesenteric glands.

2. ***The Omenta:*** There are two, the greater omentum or gastrocolic omentum, and the lesser omentum or gastrosplenic *omentum*

The *greater omentum* is the largest peritoneal fold. It consists of a double sheet of peritoneum folded on itself; therefore it has four layers It is attached to the stomach, the upper part of duodenum, and the transverse colon. It descends apronlike and unattached down the abdomen in front of the small intestine, often as far down as the pelvis It contains fat and carries the gastroepiploic arteries.

The *lesser omentum* is a duplicature of peritoneum which extends from the lesser curvature of the stomach to the liver; it encloses the hepatic vessels and ducts The esophagus lies between its two layers and its left border. At its right border, the two layers become continuous, form a free margin, and enclose the portal vein, the hepatic artery, the biliary duct, the lymphatics, and the hepatic flexure of nerves Behind this free margin and its contained vessels in front of the ascending vena cava and below the spigelian lobe of the liver is *found the* foramen of Winslow (epiploic foramen)

3 ***Ligaments:*** The ligaments are reflections of peritoneum from the walls of the abdomen or from the pelvis to the viscera. These ligaments are the hepatogastric ligament and *hepatoduodenal ligament*; they are portions of the lesser

omentum stretching from the liver to the stomach and from the liver to the duodenum respectively. The phrenocolic ligament goes from the left colic flexure to the diaphragm; it supports the spleen and is also known as sustentaculum lienis Other ligaments support such organs as the uterus and the bladder.

Peritoneal Spaces: In health the peritoneal cavity is only a potential cavity, because the abdominal contents are so crowded that there is little space between the parietal and visceral surfaces. In certain parts of the abdominal cavity, there are recesses of peritoneum forming free spaces. In health these communicate with one another; but in the presence of infection, they may become walled off as separate compartments by the mesentery or the omentum, thus preventing rapid or general extension of the disease process to other regions of the abdomen.

The major compartments are the lesser and the greater peritoneal cavities; these communicate through the foramen of Winslow Other spaces are the subphrenic, the central, the right and left lumbar, and the pelvic spaces.

Diseases of the Peritoneum

Affections of the peritoneum are classified under the general term of "peritonitis." This disease may be caused by bacterial infection, malignant invasion, or by certain systemic conditions

Peritonitis

This denotes an inflammation of the peritoneum It may run an acute, subacute, or chronic course; and it may be local, spreading, or general, depending upon the type of invading organism, its virulence, and the susceptibility of the patient

Bacteriology: Nearly all types of peritonitis, with the exception of the neoplastic and the allergic types, are caused by pathogenic micro-organisms that have gained entrance into the peritoneal cavity. The more commonly found organisms are colon bacilli, streptococci, staphylococci, bacilli pyocyanei, pneumococci, gonococci, Mycobacteria tuberulosis, and some of the anaerobic organisms. Several other types of organisms may occasionally be causative factors of this disease.

Etiology: The pathogenic organisms gain entrance into the peritoneal cavity by one of six routes:

1. The most common route is by way of a diseased abdominal viscus, i e. ruptured, inflamed, or gangrenous appendix; perforated intestine, stomach, or gallbladder; extension of infection from an inflamed viscus, such as the pancreas, uterus, or fallopian tubes, intestinal obstruction, and strangulated hernia.

2 By direct introduction of infection into the peritoneal cavity by laparotomy, stab wound, paracentesis abdominis, or by a foreign body left within the peritoneal cavity

3 By external trauma which may devitalize any of the internal organs, causing gangrene or suppuration, or cause rupture of the urinary bladder, a portion of intestines, or a gravid uterus.

4 By rupture of an intraperitoneal abscess, such as amebic, hepatic, subdiaphragmatic, or pelvic.

5 By rupture of an extraperitoneal abscess, such as supradiaphragmatic, renal, adrenal, pancreatic, or vertebral

6. By way of the blood stream or lymphatics. This may occur idiopathically or during the course of a general infection, such as pneumococcic pneumonia, gonorrhea, or subacute endocarditis.

Diagnosis: The diagnosis is based on the history of the onset, the clinical course, and the physical findings

Prognosis: Unless the spread of the infection is stopped, general peritonitis will supervene.

Acute General Peritonitis (Secondary)

This is secondary to acute local or spreading peritonitis The transition from acute local, or spreading, peritonitis to acute general peritonitis may be only a few hours or several days, depending upon the virulence of the infection. The inflammatory process involves an extensive portion of the peritoneum.

Bacteriology: Many micro-organisms are capable of producing acute general peritonitis. The most frequent offenders are colon bacilli, various types of streptococci, staphylococci, some gram-negative diplococci, gonococci, and some of the anaerobes Various other organisms have occasionally been found to be the cause of general peritonitis.

Symptoms: These vary in their severity, depending upon the character of the onset If secondary to local peritonitis, due to an inflammatory process of the adjacent viscera, the onset will be comparatively slow. If caused by a sudden rupture of a fulminating abscess, or if the pyogenic organism gains entrance into the peritoneal cavity in large numbers, the onset is sudden and the clinical picture unfolds itself within a few hours Extreme restlessness is one of the most characteristic signs of nearly all forms of peritonitis

Local Symptoms: *Pain* This is the most prominent local symptom of peritonitis. It is felt over the entire abdomen, but is most severe over the site of the initial infection Pain that is most severe in the epigastrium and is referred to the back and shoulder usually indicates a ruptured gastric ulcer. A ruptured appendix usually causes more intense pain in the right lower abdomen, but occasionally it may be referred towards the upper right abdomen. The intensity of the pain is not always an indication of the severity of the peritonitis The pain in peritonitis is continuous, though in asthenic patients it may be intermittent or remittent

Abdominal Distention: The abdomen becomes distended and is tympanitic within twenty-four hours of the onset of the disease.

Abdominal Rigidity: The abdominal muscles become rigid, and the abdominal movements are restricted so that deep breathing is not possible

Abdominal Tenderness: This is usually quite marked over the greater portion of the abdomen, and is most prominent over a local area.

General Symptoms: *Hiccough:* This occurs early in the disease and may recur at frequent intervals

Vomiting This may be more or less continuous and is aggravated by taking food, or by increased peristalsis The vomitus is at first the stomach contents; later it is brownish in color and has a fetid odor Constipation is fairly constant. Occasionally there may be diarrhea This is more common in peritonitis caused by the pneucococcus or other septic infection of the intestinal mucosa.

Temperature: The temperature depends upon the type of infecting organism. In colon bacillus infection the temperature may range from 99° F. to 101° F. In most other forms of acute peritonitis, the temperature seldom rises above 102° F. A temperature of 104° or 105° F. is usually due to an acute puru-

lent process outside of the peritoneum. The fever may be ushered in with a severe chill or with repeated chilly sensations. The temperature drops to a low subnormal level; the skin becomes cold and clammy; the pulse is weak and rapid (shock); and the patient has an anxious expression The symptoms of shock subside within a few hours, and the temperature rises

Mental State. The mental state, notwithstanding great anxiety, remains clear throughout the illness, except in the terminal stages, when mild delirium occurs, especially during the night. A statement by the patient late in the disease that he feels comparatively well, except for the abdominal pain, is a bad prognostic omen.

Physical Signs: ***Inspection:*** The patient lies in the dorsal rigid position with knee flexed. The abdomen is symmetrically enlarged and is immobile. Respirations are hurried and shallow.

Palpation: The abdominal wall is rigid, and universally markedly tender. The costal angles are flaring, and the liver and spleen are not palpable

Percussion: This should be carried out cautiously, since it induces great pain. The percussion note is tympanitic throughout unless there is an accumulation of fluid. Massive ascites is rare in acute peritonitis (See: Ascites, p 6751)

Auscultation: In the presence of a paralytic ileus, peristaltic sounds are absent, though a metallic tingling may be heard over the distended bowel. Late in the disease, when intestinal and omental adhesions are formed, cardiac sounds may be heard over the midabdomen These may be heart sounds transmitted through the adhesions or aortic sounds caused by an aorta, partially constricted by visceral adhesions

Laboratory Findings: ***Urine:*** Indicanuria is common. Albuminuria develops frequently as also do various tube casts The quantity of urine is diminished and is of high specific gravity.

Blood: The leukocyte count may range from 15,000 to 30,000 per cm. The neutrophils may rise from 80 to 90 per cent with a special increase in the young forms. In severe infections there may be a leukopenia.

Diagnosis: The cardinal symptoms of acute diffuse peritonitis are extreme restlessness, abdominal pain, tenderness rigidity, distention, tympanitis, and anxious facial expression often bearing hippocratic facies The eyes are clear; the upper lip is drawn up exposing the upper denture. The mind is clear. The temperature is elevated The blood count reveals a leukocytosis with an increase of neutrophils.

Differential Diagnosis: Acute general peritonitis is to be differentiated from lead poisoning tabetic crisis, dissecting aneurysm, acute pancreatitis, coronary occlusion, angina pectoris, intestinal obstruction, and ascites

Primary or Idiopathic Peritonitis

This condition is rare, and its diagnosis during the early stages of the disease is difficult It occurs chiefly among children, more often among girls than boys, and is seldom found in adults

Etiology: Two types of organisms have been identified with this disease, pneumococci, most prevalent in girls, and hemolytic streptococci found in both sexes. The portal of entrance has not been definitely determined. It appears that pneumococci may enter the peritoneum by way of the fallopian tubes Both types of organism probably gain entrance into the peritoneal cavity by way of the bloodstream or lymphatics.

Diagnosis: The diagnosis is based on the history of the onset, the clinical course, and the physical findings.

Prognosis: Unless the spread of the infection is stopped, general peritonitis will supervene.

Acute General Peritonitis (Secondary)

This is secondary to acute local or spreading peritonitis The transition from acute local, or spreading, peritonitis to acute general peritonitis may be only a few hours or several days, depending upon the virulence of the infection. The inflammatory process involves an extensive portion of the peritoneum.

Bacteriology: Many micro-organisms are capable of producing acute general peritonitis. The most frequent offenders are colon bacilli, various types of streptococci, staphylococci, some gram-negative diplococci, gonococci, and some of the anaerobes Various other organisms have occasionally been found to be the cause of general peritonitis.

Symptoms: These vary in their severity, depending upon the character of the onset. If secondary to local peritonitis, due to an inflammatory process of the adjacent viscera, the onset will be comparatively slow If caused by a sudden rupture of a fulminating abscess, or if the pyogenic organism gains entrance into the peritoneal cavity in large numbers, the onset is sudden and the clinical picture unfolds itself within a few hours Extreme restlessness is one of the most characteristic signs of nearly all forms of peritonitis.

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Abdominal Distention The abdomen becomes distended and is tympanitic within twenty-four hours of the onset of the disease

Abdominal Rigidity. The abdominal muscles become rigid, and the abdominal movements are restricted so that deep breathing is not possible.

Abdominal Tenderness This is usually quite marked over the greater portion of the abdomen, and is most prominent over a local area

General Symptoms: *Hiccough:* This occurs early in the disease and may recur at frequent intervals

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Palpation: The abdominal wall is rigid, and universally markedly tender. The costal angles are flaring, and the liver and spleen are not palpable.

Percussion: This should be carried out cautiously, since it induces great pain. The percussion note is tympanitic throughout unless there is an accumulation of fluid Massive ascites is rare in acute peritonitis. (See Ascites, p. 675 l.)

Auscultation: In the presence of a paralytic ileus, peristaltic sounds are absent, though a metallic tingling may be heard over the distended bowel Late in the disease, when intestinal and omental adhesions are formed, cardiac sounds may be heard over the midabdomen. These may be heart sounds transmitted through the adhesions or aortic sounds caused by an aorta, partially constricted by visceral adhesions

Laboratory Findings: ***Urine:*** Indicanuria is common. Albuminuria develops frequently as also do various tube casts. The quantity of urine is diminished and is of high specific gravity.

Blood: The leukocyte count may range from 15,000 to 30,000 per cm. The neutrophils may rise from 80 to 90 per cent with a special increase in the young forms. In severe infections there may be a leukopenia

Diagnosis: The cardinal symptoms of acute diffuse peritonitis are extreme restlessness, abdominal pain, tenderness, rigidity, distention, tympanitis, and anxious facial expression often bearing hippocratic facies. The eyes are clear; the upper lip is drawn up exposing the upper denture The mind is clear. The temperature is elevated The blood count reveals a leukocytosis with an increase of neutrophils.

Differential Diagnosis: Acute general peritonitis is to be differentiated from lead poisoning tabetic crisis, dissecting aneurysm, acute pancreatitis, coronary occlusion, angina pectoris, intestinal obstruction, and ascites

Primary or Idiopathic Peritonitis

This condition is rare, and its diagnosis during the early stages of the disease is difficult It occurs chiefly among children, more often among girls than boys, and is seldom found in adults.

Etiology: Two types of organisms have been identified with this disease, pneumococci, most prevalent in girls, and hemolytic streptococci found in both sexes. The portal of entrance has not been definitely determined It appears that pneumococci may enter the peritoneum by way of the fallopian tubes Both types of organism probably gain entrance into the peritoneal cavity by way of the bloodstream or lymphatics.

Diagnosis: The diagnosis is based on the history of the onset, the clinical course, and the physical findings.

Prognosis: Unless the spread of the infection is stopped, general peritonitis will supervene

Acute General Peritonitis (Secondary)

This is secondary to acute local or spreading peritonitis The transition from acute local, or spreading, peritonitis to acute general peritonitis may be only a few hours or several days, depending upon the virulence of the infection. The inflammatory process involves an extensive portion of the peritoneum.

Bacteriology: Many micro-organisms are capable of producing acute general peritonitis. The most frequent offenders are colon bacilli, various types of streptococci, staphylococci, some gram-negative diplococci, gonococci, and some of the anaerobes. Various other organisms have occasionally been found to be the cause of general peritonitis

Symptoms: These vary in their severity, depending upon the character of the onset. If secondary to local peritonitis, due to an inflammatory process of the adjacent viscera, the onset will be comparatively slow. If caused by a sudden rupture of a fulminating abscess, or if the pyogenic organism gains entrance into the peritoneal cavity in large numbers, the onset is sudden and the clinical picture unfolds itself within a few hours Extreme restlessness is one of the most characteristic signs of nearly all forms of peritonitis

Local Symptoms: *Pain* This is the most prominent local symptom of peritonitis. It is felt over the entire abdomen, but is most severe over the site of the initial infection. Pain that is most severe in the epigastrium and is referred to the back and shoulder usually indicates a ruptured gastric ulcer. A ruptured appendix usually causes more intense pain in the right lower abdomen, but occasionally it may be referred towards the upper right abdomen The intensity of the pain is not always an indication of the severity of the peritonitis The pain in peritonitis is continuous, though in asthenic patients it may be intermittent or remittent.

Abdominal Distention. The abdomen becomes distended and is tympanitic within twenty-four hours of the onset of the disease.

Abdominal Rigidity: The abdominal muscles become rigid, and the abdominal movements are restricted so that deep breathing is not possible

Abdominal Tenderness This is usually quite marked over the greater portion of the abdomen, and is most prominent over a local area.

General Symptoms: *Hiccough* This occurs early in the disease and may recur at frequent intervals.

Vomiting This may be more or less continuous and is aggravated by taking food, or by increased peristalsis. The vomitus is at first the stomach contents; later it is brownish in color and has a fetid odor. Constipation is fairly constant. Occasionally there may be diarrhea. This is more common in peritonitis caused by the pneucococcus or other septic infection of the intestinal mucosa

Temperature: The temperature depends upon the type of infecting organism. In colon bacillus infection the temperature may range from 99° F. to 101° F In most other forms of acute peritonitis, the temperature seldom rises above 102° F. A temperature of 104° or 105° F. is usually due to an acute puru-

lent process outside of the peritoneum. The fever may be ushered in with a severe chill or with repeated chilly sensations The temperature drops to a low subnormal level; the skin becomes cold and clammy; the pulse is weak and rapid (shock); and the patient has an anxious expression The symptoms of shock subside within a few hours, and the temperature rises

Mental State The mental state, notwithstanding great anxiety, remains clear throughout the illness, except in the terminal stages, when mild delirium occurs, especially during the night A statement by the patient late in the disease that he feels comparatively well except for the abdominal pain, is a bad prognostic omen

Physical Signs: ***Inspection:*** The patient lies in the dorsal rigid position with knee flexed. The abdomen is symmetrically enlarged and is immobile. Respirations are hurried and shallow

Palpation: The abdominal wall is rigid, and universally markedly tender The costal angles are flaring, and the liver and spleen are not palpable

Percussion: *This should be carried* out cautiously, since it induces great pain The percussion note is tympanitic throughout unless there is an accumulation of fluid Massive ascites is rare in acute peritonitis (See Ascites, p 6751)

Auscultation: In the presence of a *paralytic ileus, peristaltic sounds* are absent, though a metallic tingling may be heard over the distended bowel Late in the disease, when intestinal and omental adhesions are formed, cardiac sounds may be heard over the midabdomen These may be heart sounds transmitted through the adhesions or aortic sounds caused by an aorta, partially constricted by visceral adhesions.

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This condition is rare, and its diagnosis during the early stages of the disease is difficult. It occurs chiefly among children, more often among girls than boys, and is seldom found in adults

Etiology: *Two types of organisms* have been identified with this disease, pneumococci, most prevalent in girls, and hemolytic streptococci found in both sexes. The portal of entrance has not been definitely determined It appears that pneumococci may enter the peritoneum *by way of the fallopian tubes* Both types of organism probably gain entrance into the peritoneal cavity by way of the bloodstream or lymphatics

Symptomatology: Preceding the signs of peritonitis, there is usually a history of an upper respiratory infection and of diarrhea. *The onset is sudden, often with convulsions,* and there is generalized abdominal pain and tenderness. The child appears ill, and is restless, fretful, and vomits copiously. The temperature may reach 104° to 105° F. The leukocyte count may be between 20,000 and 30,000 with 80 to 90 per cent of neutrophils. The urine is scanty and may contain albumin and casts.

Diagnosis: Since most of the acute infections in children have a similar onset, the diagnosis of peritonitis is often overlooked. However, with a history of an acute respiratory infection, diarrhea, generalized abdominal pain, and other signs of peritoneal irritation, and in the absence of other signs of pathology elsewhere, the diagnosis of acute idiopathic peritonitis should be entertained; and an abdominal puncture is justified. The presence of pathogenic organisms in the smear or culture is conclusive evidence of the pathologic process. Great caution is necessary when performing a thoracentesis abdominis in acute fulminating peritonitis. The bowel is often distended and may easily be punctured, therefore the abdominal wall, after being anesthetized, must first be nicked with a sharp bistoury. The incision is extended to the peritoneum, then a blunt trocar with its cannula is to be slowly pushed through the peritoneal membrane.

Prognosis: Until the advent of sulfonamides, and penicillin and streptomycin, the mortality was equally high whether treated surgically or expectantly. The use of the recently discovered antibiotics has greatly reduced the mortality from this disease.

Differential Diagnosis of Various Types of Acute Peritonitis

While the main clinical features of all types of peritonitis, regardless of the etiology, are similar, their clinical course and the severity of their symptoms vary with the type of the infective organism.

Pneumococcic Peritonitis: The clinical picture in this type of infection is that of a generalized pneumococcic *septicemia with severe peritoneal involvement.* It may follow pneumococcic mastoiditis, local pneumococcic abscess, pneumococcic arthritis, or pneumococcemia. In most instances it manifests itself as an acute idiopathic peritonitis of childhood, and is more prevalent among little girls than little boys. This disease occurs in a diffuse and an encysted form.

Clinical Manifestations: Diffuse Form. This is ushered in suddenly with a chill and high temperature, severe and persistent abdominal pain, and prostration. The patient is restless, has a flushed face, and is dyspneic. Vomiting occurs often. Profuse diarrhea, instead of constipation, is a fairly characteristic finding in pneumococcal peritonitis. This is accompanied by marked abdominal tenderness with rigidity and other signs of acute peritonitis.

Encysted Form: This is more common than the diffuse type. The onset of this form is as acute as in the diffuse type. However, during the next few days, the symptoms ameliorate and the patient appears improved. After this respite, the patient has a relapse, and the abdomen becomes distended, tender, and painful. The diarrhea is replaced by obstinate constipation, and signs of septicemia become manifested. At this time an abscess *is usually formed in the lower abdomen* which has a tendency to rupture at the

umbilicus. The pus is odorless and of greenish color.

Prognosis: The outcome of pneumococcic peritonitis is extremely doubtful; however, the sulfonamides and the recently discovered antibiotics have greatly decreased its mortality.

Streptococcic Peritonitis: This may result from puerperal septicemia or it may be secondary to lesions of the alimentary canal. The onset is sudden with severe chills, high fever, restlessness, and abdominal colicky pain. The abdomen becomes ballooned out, and at first there is local tenderness which later becomes generalized. Diarrhea occurs as often as constipation The patient presents general signs of severe septicemia associated with peritonitis The history and the associated signs will help to differentiate puerperal sepsis from alimentary lesions as the cause of the peritonitis

Prognosis: Before the advent of sulfonamides and the antibiotics, this type of peritonitis was fatal in the great majority of cases Since the use of these drugs, the mortality rate has been greatly reduced

Gonococcic Peritonitis: This usually follows gonococcic infection of the genitals. It is found more frequently among females than among males, because of the direct connection of the fallopian tubes with the peritoneal sac. This infection may occur as an acute diffuse peritonitis or as a local pelvic infection

Clinical Manifestations: *Acute Diffuse Gonococcal Peritonitis* The onset is ushered in with a chill or, more often, with a series of chilly sensations The temperature rises to 101° F or higher, and there are intermittent sweats. The abdomen becomes tense and is tender to touch, particularly in the pelvic region. Constipation is the rule, and other signs of peritonitis become manifested In the female, the infection spreads by way of the fallopian tubes and, in the male, by way of the epididymides. In both sexes the rupture of a pelvic gonococcic abscess may cause the peritoneal infection. The organisms found in gonococcic peritonitis are usually a mixture of gonococci and colon bacilli.

Local Gonococcic Peritonitis: As its name indicates, it is a peritoneal inflammation localized in the pelvis; it often results in an abscess. The onset is abrupt with a series of chills, fever, and some sweats, and distention of the lower abdomen with exquisite tenderness in the lower pelvis. There is marked constipation, and often difficulty in passing urine or urinary retention. This is associated with other signs of peritoneal irritation The diagnosis is made on the history of the primary infection, a pelvic examination, and a positive complement-fixation test for gonococci.

Prognosis: In both the diffuse and localized forms of gonococcal peritonitis, the prognosis is good. Patients usually recover. However, pelvic or general peritoneal adhesions may remain as sequelae of this infection.

Chronic Peritonitis

Chronic peritonitis may be localized or diffused

Chronic Localized Peritonitis (Peritoneal Adhesions): This may result from an attack of acute local peritonitis, may follow a laparotomy or a stab wound in the abdomen, or it may be nature's method of walling off perforated or inflamed abdominal viscera. Occasionally local adhesive bands may be congenital.

Localized adhesions in the right upper abdomen (hepatic flexure) may result

from cholecystitis or from acute or subacute perforation of a duodenal or gastric ulcer. Intestinal adhesions may be formed because of diverticulitis of the ileac and pelvic colon, or occasionally because of ulcerative colitis. Adhesions in the lower right quadrant may be caused by acute or chronic appendicitis or oophoritis, particularly if appendectomy or oophorectomy required drainage Pelvic adhesions may result from pelvic inflammation and affect the appendix, ovaries, pelvic colon, cecum, sigmoid, or rectum. Local chronic peritonitis may also be caused by peritoneal tuberculosis or by gonococcal peritonitis.

Congenital local adhesions with the formation of narrowing or kinking, causing partial obstruction of the bowel, is seen in the so-called "Lane's kink" (kinking of the lower end of the ileum due to adhesions).

Chronic Diffuse Peritonitis: This may be due to tuberculosis or to malignancy as primary causes (See: pp. 675i, 675k) It may also occur as a secondary manifestation of chronic disease elsewhere in the body, or it may be the aftermath of an acute peritonitis Chronic peritonitis is more often regional than diffuse. The peritoneum becomes thickened, loses its luster, and adhesions may form around the intestines, omentum, and other abdominal organs. In some instances, a serofibrinous exudate may form. In Pick's syndrome (*pericarditic pseudocirrhosis*), both the visceral and parietal surfaces of the peritoneum are involved, and ascites is a prominent symptom. In *actinomycosis*, echinococcus cysts may form in any part of the peritoneum and form abdominal tumors.

Symptoms: The symptoms of chronic peritonitis depend upon the primary cause and upon the degree of its interference with visceral function. Abdominal pain is usually colicky; it may be more or less constant, or it may occur periodically. The pain is increased during active peristalsis; it may occur before, during, or after a bowel movement. There is a sense of fulness, dragging, or spasticity in the abdomen or perineum. The appetite is poor and digestion is interfered with There may be diarrhea or constipation The abdomen is usually distended, and tenderness may be elicited over various parts of the abdomen. The liver may be enlarged, particularly in perihepatitis. Abdominal pulsation may be palpable and audible. The patient is chronically ill, often loses weight, and may run a subfebrile temperature. Enlarged abdominal veins are often present, and ascites may develop when the intra-abdominal circulation is interfered with.

Tuberculous Peritonitis

Tuberculous peritonitis is a disease of childhood and early adulthood The primary focus may be in the mesenteric glands, the lungs, the prostate, the seminal vesicles or testes, the fallopian tubes, the intestines, or the kidneys In older persons, as well as in the young, generalized tuberculous peritonitis may be an accompaniment of acute miliary tuberculosis. The infection may spread by way of the lymphatics, in which case it is usually more or less localized, or by the bloodstream, in which case it is generalized (miliary) Tuberculous peritonitis may be acute or chronic.

Acute Tuberculous Peritonitis: This occurs as a general widespread affection invading nearly all the tissues of the body.

Symptoms: The clinical manifestations are those of any acute infection There is high fever, sweats, rapid pulse,

rapid breathing, diarrhea, abdominal distention, and usually ascites. Other local manifestations depend upon which of the structures are most seriously involved. When the brain and the meninges are copiously infiltrated, nervous symptoms will be in evidence. If the lungs or pericardium are severely affected, there will be marked pulmonary or cardiac signs. When the liver or biliary system are affected, ascites or jaundice may be prominent factors.

Pathology: The lesions are spread over large areas as grayish tubercles of various sizes which eventually coalesce and caseate

Diagnosis: The presence of high fever, sweats, signs of peritoneal involvement, associated with either neurologic or respiratory symptoms, and the finding of choroid tubercles are suggestive of tuberculous peritonitis The diagnosis may be confirmed by an x-ray study of the lungs, a spinal puncture, and a positive tuberculin test

Chronic Tuberculous Peritonitis: This occurs in three main clinical forms (1) The ascitic; (2) the caseous or loculated, and (3) the obliterative, adhesive, or fibroid. These forms depend on the severity of the infection and the resistance of the patient.

Symptoms: The general symptoms of chronic tuberculous peritonitis may at times remain latent for extended periods Preceding the onset of the abdominal symptoms, there is a history of loss of weight and strength, and of general ill health stretching over a period of many months During that time there is a slight evening rise of temperature, some night sweats, loss of appetite, constipation or diarrhea, or constipation alternating with diarrhea. The stools are sometimes bulky and pale. Ascites is present in type 1 and to a lesser degree in type 2.

Physical Signs: The patient is emaciated. The skin is dry The abdomen is distended, and its skin covering is dark brownish, resembling Addison's disease. On palpating the abdomen, there is a peculiar unelastic, doughy, dead rubbery resistance. The spleen and, at times, the liver are palpable.

Diagnosis: *Ascitic Form:* This is common in children and adolescent girls

Symptoms and Physical Signs: Since the exudate develops gradually, there is, at first, tympany which later gives way to dulness The amount of fluid in the abdomen is as a rule not large. Occasionally there may be a rapid accumulation of a fairly large amount of fluid which stretches the abdominal wall causing great pain, and when the vena cava becomes partially compressed edema of the legs develops. The ascitic fluid is generally clear, contains large numbers of lymphocytes, and often coagulates on standing. Mycobacteria tuberculosis are seldom cultured from the fluid; however, when this is injected into a guinea pig, it will produce tuberculosis.

The ascitic form may be acute, subacute, or chronic The acute form differs from the chronic in that the miliary tubercles are small and are scattered over the whole peritoneum. Adhesions are seldom formed. In the chronic type, the tubercles are large and fibrotic, the peritoneum is thickened, and the mesentery is shortened, thus confining the intestines to the posterior abdominal wall Therefore, on percussion, dulness is often elicited over the anterior abdominal wall, and resonance in the lumbar regions Change of posture does not change the areas of dulness or resonance.

Caseous or Loculated Form. Loculated areas containing fluid are formed by matted intestinal coils, masses of tuberculous material, and greatly thickened omentum. During the early stages, the encysted fluid is clear, but later becomes purulent and may break through the umbilicus, the vagina, or into a coil of intestine.

SYMPTOMS: The patient is pale and emaciated, the face is generally flushed, and the eyes appear clear. There is usually a subfebrile temperature with evening exacerbations and night sweats. The pulse is rapid. Colicky pain may be constant or it may occur before, during, or after defecation or vomiting. There may be obstinate constipation or profuse diarrhea; frequently they alternate.

PHYSICAL SIGNS The abdomen is greatly distended and may appear irregular. The skin over the abdomen is stretched and is dry and pigmented Palpation over the abdomen imparts a doughy, unelastic resistance, and various sized and shaped masses may be felt in the abdomen. Tenderness is not marked Percussion will elicit irregular areas of tympany and dulness, depending on the distribution of entrapped, distended knuckles of intestines and the caseated or matted material Auscultation reveals that peristaltic sounds are weak; often they are absent over various regions. Cardiac or aortic pulsations may be heard as low as the umbilicus or lower, and at times they may also be heard in the flanks. (*Caution:* The abdomen should not be tapped in such cases.) A search should be made for tuberculous foci elsewhere. A tuberculin test, while not conclusive when present in an adult, is nevertheless worth doing. X-ray study of the intestines may reveal irregular areas of constriction of the intestinal lumen. Tuberculous ulcerations of the colon may also be discovered thus.

Obliterative, Adhesive, or Fibroid Form: This may be a healing process, occurring after the absorption of fluid in the ascitic form, or it may be a primary process. There is a widespread formation of adhesions binding down all the abdominal viscera to the parietal peritoneum and causing symptoms of partial intestinal obstruction. Ascites is absent.

SYMPTOMS· These are ill-defined. The patient is usually weak, emaciated, and irritable. Abdominal pain is often severe, particularly prior to, or during, defecation The abdomen is somewhat distended. Palpation imparts a doughy, rubbery resistance, and irregular masses may be palpable On percussion, tympany and dulness are elicitable over irregular areas of the abdomen On auscultation, peristaltic sounds are marked, and pulsation is audible over the upper part of the abdomen.

MALIGNANT PERITONITIS (CANCER OF THE PERITONEUM)

This is nearly always secondary to cancer of the abdominal viscera or other structures in the body. The primary lesion may be in the stomach, liver, pancreas, intestines, ovary, uterus, breast, testes, prostate, or elsewhere.

Symptoms: In most instances, the symptoms of the primary focus overshadow the peritoneal manifestations except when ascites is present.

Physical Signs: The abdomen is distended. Colicky pain and acute lancinating pain are felt over various parts of the abdomen, particularly when the spinal roots or peritoneal nerves are involved In the absence of ascites, when the cancerous masses are large or when they cause matting of the bowel, mesentery, or

omentum, they may be felt through the abdominal wall. The umbilicus is often infiltrated with cancerous growth. Ascites, when present, is easily discoverable by physical examination (See. Ascites, below) The ascitic fluid may be clear, turbid, hemorrhagic, chyliform, chylous, or icteric and may contain cancer cells

A laparotomy will yield a positive diagnosis and may, at the same time, be the means of retarding the infection.

Peritonitis of Systemic Origin

There are reported in the literature rare instances of peritonitis with ascites not caused by any local or intra-abdominal infection It is questionable whether these conditions should be classified as true peritonitis or as peritoneal irritation occurring in conjunction with other symptoms during the course of certain diseases, particularly so since the peritoneal involvement disappears when the systemic affections clear up

Allergic Peritonitis

Sison[1] and co-workers reported the case of a young woman who developed recurrent attacks of peritonitis associated with pregnancy One attack occurred during her sixth month of pregnancy, and three attacks occurred from two to four weeks postpartum Another, though milder attack, was induced by the administration of 1000 units of estrone subcutaneously, this was later controlled by the administration of progesterone The patient and several members of her family had a history of rheumatic fever, and there was a strong familial and personal history of allergy.

The clinical features, during the attacks of peritonitis, were severe epigastric pain radiating over the entire abdomen, accompanied by abdominal tenderness and ragidity and by vomiting. There was a moderate amount of ascitic fluid which contained a high cell count, high albumin percentage, high specific gravity, and high eosinophilic count (38 per cent). The blood count revealed a leukocytosis of 30,000 with 38 per cent of eosinophils. Laparotomy failed to reveal any definite intra-abdominal disease. The patient made a complete recovery after each attack, and remained well during the interims of her pregnancies, but the symptoms recurred at the conclusion of each pregnancy. Since this period corresponds to the time of greatest estrogen production, the authors believed that their patient's peritonitis was an allergic reaction to that hormone.

Ascites (Intraperitoneal Dropsy, Abdominal Dropsy)

Definition: By ascites is meant the presence of free fluid within the peritoneal cavity.

General Consideration: Ascites is a symptom present in a number of diseases which cause an accumulation of various quantities of free fluid within the peritoneal sac. The peritoneal cavity normally contains between 100 and 200 cc. of clear fluid, not recoverable by tapping The amount is constant, since the rates of formation and absorption are equal.

Conditions Causing Ascites: (1) Excessive outpouring of fluids within the peritoneal cavity; (2) diminished absorptive capacity of the peritoneum; and (3) both increased production and decreased absorption of intraperitoneal fluid

1. ***Excessive Outpouring of Fluid Within the Peritoneal Cavity:*** This may be due to the following:

[1] Sison, A, Dionisio, S A, Silva, J. A, and Chavez, P. C. · J A M A 134 1007, July, 1947

(*a*) Hypoproteinuria (See p. 1011). A decrease in the plasma albumin irrespective of its cause will, according to Trumper and Cantarow, result in diminished plasma colloid osmotic pressure within the blood vessels. This decreases the ability of the plasma to hold water, *thus causing an extravasation of water* into the tissues.

(*b*) Venous obstruction. Obstruction of the portal vein or of any other large intra-abdominal vessel from within (thrombosis) or pressure from without (extrinsic).

(*c*) Increased capillary permeability

(*d*) Peritoneal inflammation.

(*e*) Rupture of an aneurysm or other blood vessel

(*f*) Interference with the flow of lymph through the mesenteric lymphatics, the receptaculum chyli, or the thoracic duct, and rupture of the thoracic duct or lymphatic vessels.

2 ***Diminished Absorptive Capacity of the Peritoneum:*** This may be caused by:

(*a*) Decreased intraperitoneal tension.

(*b*) Slowing of the peritoneal circulation.

(*c*) Peritoneal edema

(*d*) High protein content of ascitic fluid (exudate, pus, blood).

3. ***Increased Production and Decreased Absorption of Intraperitoneal Fluid:*** This may be caused by·

(*a*) Interference with the venous return of the peritoneal cavity.

(*b*) Interference with the portal circulation either within the peritoneal cavity or within the liver.

(*c*) Increased venous pressure within the hepatic veins, the inferior vena cava, or the right side of the heart

(*d*) Increased intracapillary pressure within the peritoneal cavity

Quantity: The quantity of accumulated fluid within the peritoneal cavity varies It may measure several gallons or a quart or two When the effusion is large, it may fill the entire abdominal cavity, enormously distend the abdomen, displace the abdominal viscera, and partially compress the thoracic organs. When the effusion is small, it may cause little if any inconvenience, unless its underlying cause is inflammation, hemorrhage, a ruptured viscus, or malignancy.

Character of Ascitic Fluid: There are seven types of fluid recoverable by paracentesis abdominis· (1) Transudates; (2) exudates; (3) purulent; (4) chylous; (5) pseudochylous; (6) sanguineous; and (7) icteric.

1 ***Transudates:*** *Appearance*. Greenish, straw-colored, or colorless. *Specific gravity* Below 1 015 *Protein content·* 1 to 3 per cent *Rivalta test* shows a faint bluish or smoky suspended cloud. *Cytology* Few endothelial cells and occasional white cells. *Odor* Not offensive

This type of fluid is found in cirrhosis of the liver, nephrosis, nephritis, hypoproteinuria, ulcerative colitis, severe anemia, portal vein compression, cardiac decompensation, and most of the other noninflammatory conditions In cardiac decompensation, the fluid has a specific gravity of 2 to 3 per cent, clots spontaneously, and contains endothelial cells and lymphocytes

2 ***Exudates:*** *Appearance:* Cloudy. *Specific gravity·* Above 1 015. *Protein content.* 3 to 5 per cent or above *Rivalta test* shows a bluish precipitate falling to the bottom of the test tube *Cytology* Polymorphonuclear leukocytes, lymphocytes, and red and white blood cells, depending upon the degree of peritoneal inflammation

Exudates are the product of inflamma-

tion and are found in peritonitis, tuberculosis, and malignancy. In tuberculosis, the predominating cell content is the lymphocyte In cancer, cancer cells and bits of cancer tissue may be recovered in a centrifuged specimen. Various bacteria, such as streptococci, staphylococci, colon bacilli, and Mycobacteria tuberculosis may be obtained by culture, if infection of the peritoneum is due to these organisms.

3. ***Purulent Fluid:*** *Appearance*· Turbid. *Specific gravity* Above 1.015. *Protein content:* High. *Odor:* May be offensive. *Cytology* Polymorphonuclear cells, red and other white blood cells, and various micro-organisms may be discovered by smear or culture.

This is found in suppurative conditions of the peritoneum which may result from a ruptured appendix or pus tube, or from empyema of the gallbladder, abscessed liver, suppurative pancreatitis, or perforation of the bowel or stomach.

4 ***Chylous Fluid:*** *Appearance:* Milky. *Specific gravity:* 1.015 to 1.040. *Protein content:* Above 3 per cent. It does not clot but separates into layers on standing. It contains fine fat globules and glucose. This is a rare condition which may be caused by infestation of the lymphatics by Wuchereria bancrofti (Falaria sanguinis-hominis nocturia), and may also be due to rupture of the receptaculum chyli or other lymph vessels. Occasionally it may be found in tuberculous or malignant disease. An excessive fat diet may at times cause an existing transudate to become slightly chylous.

5 ***Pseudochylous Fluid:*** This may be of two types. One type contains large fat globules which are caused by fatty degeneration of large numbers of newly formed cells or old leukocytes. The other type of chyloform ascitic fluid does not contain fat globules. Its milky appearance is due to some form of lecithin or globulin and may be associated with lipemia. This condition may also be found in malignant or tuberculous peritonitis, and occasionally occurs in some cases of nephrosis.

6 ***Sanguineous Fluid:*** This is usually caused by rupture of an aneurysm, extrauterine pregnancy, graafian follicle or corpus luteum, or by rupture of a blood vessel. It may also be caused by purpura and other blood dyscrasias, or by a degenerating malignant tumor.

7. ***Icteric Fluid:*** Bile-stained fluid may be found in any condition causing ascites when associated with jaundice and also when there is an associated rupture of the gallbladder or bile duct. In acquired tertiary syphilis of the liver, the ascitic fluid may be hemorrhagic or icteric In carcinoma of the liver, the ascitic fluid may be both hemorrhagic and icteric.

Diagnosis of Ascites

History: It is important to elicit a detailed history as to the onset of the abdominal enlargement, and the nature of previous illness and of recent illness referable to the cardiovascular system, the digestive system, the urinary system, the pelvic organs, and the hemopoietic system. Inquiry should also be made as to food and drink habits, and of a possible recent or remote trauma or surgical operation

Symptoms: Other symptoms associated with ascites depend upon the diseases causing it and upon the quantity of fluid present. When the quantity of fluid is large, the symptoms will be those of pressure or crowding of the viscera, viz.: Abdominal discomfort, dyspnea, at times orthopnea, cardiac palpitation, digestive

disturbances, and weakness. If the ascites is associated with infection, there will be, in addition to signs of fluid, abdominal pain, toxic symptoms, and other signs of infection.

Physical Signs: ***Inspection:*** In the presence of a large effusion, with the patient lying on his back, the abdomen is symmetrically enlarged and there is definite bulging in both flanks, resembling a frog's belly. The skin over the abdomen is tense, and appears thin and often glistening. The superficial veins are prominent and appear as blue streaks; at times they are elevated and, in rare instances, appear concentrated in the umbilical region (caput medusae). The umbilicus is flattened or bulging. In old cases, atrophic lines (lineae atrophicae) develop in the flanks. The respiratory movements are of the supracostal type. Abdominal movements are absent. The diaphragm is elevated. The liver and the apical impulse are displaced upwards.

Palpation: A uniform smooth tenseness is palpable over the entire abdomen which is, however, more resistant in both flanks than in the umbilical region. Ballottement may be elicited if the fluid covers an enlarged liver, spleen, or a tumor.

Percussion: Dulness is elicitable in both flanks and resonance over the highest point in midabdomen. This is due to the floating of the intestines upon the fluid. When the intestines are bound down by adhesions, a dull note will be elicited over the entire abdomen. Shifting dulness on change of posture is an important sign of ascites. It is elicitable when the amount of free fluid in the abdomen exceeds three pints (1500 cc.). Change of posture will change the areas of dulness and resonance, so that dulness will be found in the dependent parts and resonance in the uppermost parts of the abdomen. In the knee-chest position, resonance may be elicited along both sides of the spinal column and dulness in the umbilical region. The upper area of liver dulness may reach as high as the third or fourth rib. This should not be mistaken for pleural effusion. To differentiate between a liver pushed up by ascites and pleural effusion, the following is to be noted. In ascites, when the patient sits or stands, the upper line of dulness is lower than when lying supine; also, during forced inspiration, the line of dulness becomes lower. Such change in the line of dulness is absent in right-sided pleural effusion. Fluctuation is another important sign of ascites. (See Technic, p. 590.) To be pathognomonic, fluctuation plus shifting dulness must be present. Fluctuation may not be elicited when the amount of free fluid is small and the abdominal wall is obese, nor may it be elicited when the quantity of fluid is so large that it causes extreme tenseness of the abdominal wall. Occasionally fluctuation may be elicited over a large cyst. Shifting dulness in the absence of fluctuation may at times also be demonstrated in chronic obstruction of the small intestines.

A small amount of ascites in an obese person or in the presence of marked edema of the abdominal wall is often difficult to demonstrate. However, percussion dullness is more pronounced over the umbilical region, with the patient in the knee-hand position, than it is when he is in the supine position. In the Trendelenburg position, ballottement may be detected over an enlarged liver. In the sitting position, percussion over the quadrate muscles will cause a vibratory tremor over the anterior abdominal wall (Pitfield's sign).

Auscultation: This procedure is of questionable value in ascites. A metallic tinkling sound may at times be heard over the umbilical region when the abdomen is shaken. This is also heard over a greatly distended or paralyzed bowel and in intestinal obstruction. Borborygmus may be heard over the umbilical region; it is absent in the flanks.

Other Diagnostic Aids: *Rectal examination* may reveal hemorrhoids, indicating portal obstruction. The presence of carcinoma of the rectum or prostate may thus be found. *Pelvic examination* may reveal a collection of fluid in the cul-de-sac, or a tumor of the uterus or of the ovary (Meige's syndrome).

Roentgen-Ray Studies: These may reveal displaced abdominal organs, such as the bowels, the liver, and the spleen.

Peritoneoscopy: By this procedure, the abdominal organs can be visualized and a biopsy taken of any suspicious tissue, at the same time the ascitic fluid is removed. However, this procedure is not without risk of causing a ruptured viscus

Paracentesis Abdominis: (See p. 1028) While this is one of the best diagnostic procedures, nonetheless one should be reasonably certain of his diagnosis before he taps the abdomen. He may find that instead of an ascitic abdomen he has tapped an ovarian cyst or, what may be worse, a distended bowel, an overdistended bladder, or a polyhydramnios

Diseases Associated with Ascites

I. Diseases of the Liver and Portal System:

(*a*) Cirrhosis of the liver (portal, Laennec's).

(*b*) Syphilis (acquired tertiary)

(*c*) Carcinoma of the liver (primary)

(*d*) Thrombosis of the portal vein, the splenic vein, the hepatic artery.

(*e*) Mesenteric thrombosis.

(*f*) Hemochromatosis (bronzed diabetes).

(*g*) Tumors of the liver and echinococcus cyst.

(*h*) Distomatosis hepatica.

(*i*) Perihepatitis.

(*j*) Pick's disease (multiple serositis).

II. Diseases of the Spleen:

(*a*) Banti's syndrome.

(*b*) Egyptian splenomegaly (Schistosoma-mansoni infection)

(*c*) Perisplenitis.

III. Diseases of the Blood-Making Organs:

(*a*) Severe primary and secondary anemia

(*b*) Aplastic anemia.

(*c*) Leukemia (severe myeloid and lymphocytic)

IV. Diseases of the Heart and Blood Vessels:

(*a*) Cardiac decompensation

(*b*) Constrictive pericarditis.

(*c*) Pericardial effusion.

(*d*) Pulmonary embolism.

(*e*) Obstruction of the inferior vena cava (thrombosis or pressure).

V. Intra-abdominal Tumors and Enlarged Lymphatic Glands:

(*a*) Hypernephroma (invading the inferior vena cava).

(*b*) Lobstein's cancer (*retroperitoneal sarcoma*)

(*c*) Uterine tumor (benign and malignant).

(*d*) Ovarian tumors.

(*e*) Meige's syndrome (ovarian fibroma with ascites and hydrothorax). May also occur with malignant tumor of the ovary.

(*f*) Any intra-abdominal tumor or gland when sufficiently large enough to cause compression of the vena cava, the

portal veins, or the mesenteric vessels may produce ascites.

VI. Diseases of the Kidneys:

(*a*) Acute glomerular nephritis.

(*b*) Chronic nephritis (nephrotic stage).

(*c*) Lipoid nephrosis.

(*d*) Polycystic disease of the kidneys.

VII. Diseases of the Peritoneum:

(*a*) Acute local or general inflammations caused by suppurative conditions of the abdominal organs, rupture of a hollow viscus, extension from an inflamed organ, or from other causes

(*b*) Chronic adhesive peritonitis.

(*c*) Tuberculous peritonitis.

(*d*) Malignancy of the peritoneum or omentum.

(*e*) Infection of the peritoneum following a wound or surgical operation.

(*f*) Foreign bodies within the peritoneal cavity.

VIII. Diseases of the Lymphatic System:

(*a*) Lesions of the mesenteric and peritoneal lymphatics and nodes.

(*b*) Injury, disease, or obstruction of the receptaculum chyli or thoracic duct.

(*c*) Lymphomas.

(*d*) Filariasis.

IX. Hypoproteinuria:

(*a*) Nutritional deficiencies.

(*b*) Ulcerative colitis.

(*c*) Nephrosis.

(*d*) Any condition that causes depletion of the serum albumin.

X. General Wasting Diseases.

XI. Periarteritis Nodosa.

Diagnosis of Conditions Causing Ascites

Portal Cirrhosis (Laennec's Cirrhosis): The abdomen is uniformly markedly enlarged. Superficial abdominal venous enlargement is prominent. The patient is emaciated and is often slightly jaundiced. Hemorrhoids and esophageal varices are common. Vomiting, hematemesis, and digestive disturbances are frequently observed. The liver may not be palpable; and when it is felt, it is hard, irregular, and not very tender to the touch. The spleen is enlarged. The ascitic fluid is a transudate and reaccumulates rapidly after tapping. When peritonitis develops as a complication, the ascitic fluid becomes an exudate. The serum protein is low and the globulin is comparatively high in proportion to the albumin.

Syphilis of the Liver: The ascites is of moderate quantity. The fluid is often a transudate and is frequently bile-stained. The liver may be irregular in outline, not tender to touch, and the left lobe is comparatively larger than the right. Serology yields a positive reaction to syphilis

Carcinoma of the Liver (Primary): The ascitic fluid is an exudate and may be both bile-stained and bloody. The liver becomes progressively enlarged, is often tender to the touch, and is irregular in outline, frequently presenting bosselated areas having umbilicated centers.

Portal Thrombosis (Acute): There is sudden and rapidly accumulating ascites which reaccumulates rapidly after tapping. The fluid may be blood-stained, or only a transudate. Abdominal pain is persistent. The spleen is markedly enlarged. Hematemesis, melena, diarrhea, and at times ileus are prominent symptoms.

Hemochromatosis (Bronzed Diabetes): The patient has a dark bronze discoloration over the entire body. The liver when palpable appears hard. The spleen is palpable. There is marked emaciation. Hyperglycemia and glyco-

suria are marked, and the ascitic fluid, while clear, contains glucose.

Mesenteric Thrombosis: There is a history of sudden acute abdominal pain, vomiting, prostration, and melena. In the presence of a moderate effusion, the umbilicus shows evidence of a hematoma or is discolored. Abdominal paracentesis will disclose a bloody fluid.

Pick's Disease: There is a generalized edema. The liver is small and the spleen is enlarged. The fluid is a transudate. Venous pressure is high, and there is evidence of circulatory failure.

Diseases of the Spleen: With marked splenomegaly, the ascitic fluid in Banti's syndrome is a transudate. In bilharziosis, the spleen is very large, tender, and hard. The liver is enlarged. Eosinophilia with leukopenia is found in the blood smear. The ascitic fluid is often an exudate. Abdominal pain and bloody diarrhea are usually the predominating symptoms.

Diseases of the Blood-Making Organs: In severe primary and secondary anemia, the blood count is characteristic of the disease, hypoproteinuria is usually marked. The ascitic fluid is a transudate. There is generalized edema.

Cardiac Disease: In cardiac decomposition (myocarditis, tricuspid insufficiency), the liver is large and smooth. The spleen is not enlarged. There may be an accompanying pleural effusion, more often on the *left* side than the *right*. In addition to the ascites, there are present other definite symptoms of heart failure, such as dyspnea, congestion of the lungs, cyanosis, and distention of the superficial veins in the neck. The ascitic fluid coagulates on standing. Occasionally recurrent ascites may be found in organic tricuspid disease, in pericardial adhesions, or in certain cases of mitral stenosis, without any obvious evidence of cardiac insufficiency.

Intra-abdominal Tumors: Often there is a history of a gradual increase in the size of the abdomen preceding the ascites. Pelvic examination may reveal a uterine or ovarian tumor. Edema of the legs is common, and abdominal pain due to pressure may be an early symptom. In the presence of benign tumors, the fluid is a transudate.

Lobstein's Cancer (Retroperitoneal Sarcoma): This usually occurs in children, and at times in adults (more often in males) between the ages of thirty and forty. There is a history of stabbing abdominal pain frequently mistaken for appendicitis, cholecystitis, pancreatitis, or gastric or duodenal ulcer. When the tumor is larger, there is pain on pressure over the mass which is referred downwards to the lower extremities. When ascites occurs, the patient is weak, emaciated, and may have accompanying edema of the lower extremities. The liver and spleen are not enlarged. The ascitic fluid may be a transudate.

Tuberculous Peritonitis: In tuberculous peritonitis, the amount of free fluid in the abdomen is rather scant. The abdomen, on palpation, yields a peculiar doughy or dead-rubbery resistance. Fluctuation is not readily elicited. There may be scattered areas of dulness intermingled with areas of resonance. Auscultation of the abdomen will transmit the cardiac sounds, and when the abdominal aorta is partially constricted by adhesions, the aortic pulsation may be heard synchronously with the apex beat. There is usually some induration or redness of the umbilicus. The ascitic fluid is generally turbid, contains many leukocytes, and has other signs of an exudate. This condition is more common among children or young

adults. Paracentesis should be done with caution. The skin and underlying muscle, at the site of operation, *should first* be divided with a bistoury and a dull-pointed trocar should be used.

Chronic Peritonitis: The mesentery may be so shortened that it does not permit the intestines to float above the fluid, thereby causing the fluid to accumulate on top of the intestines. A dull note may be elicited when percussing the highest part of the abdomen, which in other forms of ascites yields a resonant note Percussion in the prone position will yield resonance near the spine

Periarteritis Nodosa (Rare): There is severe abdominal pain, often migratory. The temperature is elevated. Nephritis is common. Leukocytosis and marked eosinophilia are significant findings.

Ascites due to other causes can usually be diagnosed by obtaining a comprehensive history, by a careful physical examination, and by the employment of such laboratory aids that will help to ascertain its cause.

Differential Diagnosis

Conditions Simulating Ascites: 1. ***Obesity:*** This may easily resemble *ascites, particularly when associated with* myocarditis, an enlarged liver, edema of the legs, and dyspnea To differentiate obesity from ascites one should note that, in obesity, dulness is elicited uniformly over the entire abdomen, the flanks do not bulge, and shifting dulness is absent, i.e. when the patient is placed in the knee-chest position, the percussion note does not differ from that elicited when the patient lies flat on his back, nor does the change in posture from one side to the other change the quality of the percussion note.

2. ***Large Ovarian Cyst:*** This may be difficult to differentiate from ascites, *particularly in an elderly* woman who also suffers from cardiac weakness. The presence of an ovarian cyst may be diagnosed by the abdominal enlargement *which may or may not be symmetrical,* but is most prominent anteriorly below the umbilicus. The umbilicus is not flattened nor is it protruding There is tympany in the flanks, and there is no change in the area of dulness or resonance on change of posture. When placed in the Trendelenburg position there is no increase in the dyspnea, such as is found in ascites, nor is there a change in the percussion note A mass which is usually confined to the lower abdomen may be elicited by careful palpation. Vaginal examination in the dorsal position, the Trendelenburg, and then in the Walcher position will reveal a greater shift in fulness of the vaginal dome in ascites, and not in ovarian cyst. The history of onset is important. The fluid contains albumin, cholesterin crystals, and granular matter.

3. ***Hyperhydramnion:*** This may be *differentiated from ascites by the history* suggesting pregnancy, the definite line of demarcation from dulness to clearness, the presence of ballottement which may *be elicited below the umbilicus, the* characteristic findings by pelvic examination, and the confirmation of pregnancy by x-ray examination. Fetal heart sounds may be heard after the fifth month of pregnancy.

4. ***Intestinal Obstruction:*** Obstruction of the small intestines will cause dulness in the flanks and over irregular areas of the abdomen There *is an ab*sence of borborygmus, though splashing sounds may be elicited by direct percussion of the abdominal wall, due to accumulated fluid in the intestines. The

history of constipation, vomiting, and pain aid in the diagnosis of intestinal obstruction.

5. ***Overdistended Bladder:*** This condition may resemble ascites, particularly when it reaches the umbilical area. However, the mass is hard and rounded. Pressure upon it will cause pain or a sense of discomfort. There is tympany in the flanks and dulness over the anterior abdominal wall There is no change in the percussion note on change of position. When in doubt, the bladder can easily be catheterized It is less risky to insert a catheter through the urethra than to plunge a needle through the abdominal wall.

6. ***Hirschsprung's Disease:*** The abdominal distention is chiefly in the region of the colon, though the entire abdomen is enlarged. The whole abdomen may be tympanitic, and when the colon is overfilled with feces, dulness may be elicited over that part of the colon. There is no change in the percussion note on change of position. There is no tenderness on pressure, and there is usually a history of long-standing abdominal distention since early childhood.

7. ***Acute Peritonitis:*** This may resemble ascites However, the fever, rebound tenderness, generalized tympany, and other signs of severe acute infection are the differential points (See: p. 675f.)

CHAPTER XXIV

Examination and Diseases of the Urogenital System

The Kidneys

Physical Examination of the Kidneys

The Normal Kidney: *Inspection* of the surface of the body as an aid in the diagnosis of kidney conditions is not very valuable, because a kidney is seldom so large that its bulging can be noted by inspecting the kidney regions; however, in cases of sarcoma in young children, or hydro- and pyonephrosis or hypernephroma in a thin adult, a swelling may be seen in the region of the affected kidney both anteriorly and posteriorly.

To *palpate* the kidney properly, the patient should lie supine, shoulders and knees slightly elevated, the examiner slipping one hand under the back so that the index finger rests upon the lower rib and the adjoining two fingers support the soft tissue, the other hand being laid flat upon the abdomen, resting below the costal margin. The patient should be instructed to breathe deeply while the examiner attempts to approximate both of his palpating hands. If the kidney is in a low position, a soft rounded mass may be palpated. The normal kidney is seldom palpable except during forced inspiration in patients who have extremely thin and flaccid belly walls When the kidney is being pressed upon, the patient usually complains of tenderness, pain or of a "sickening feeling" or of a desire to micturate.

Outlining a normal kidney by *percussion* is not always satisfactory. If any degree of accuracy is to be obtained, percussion should be done in the flanks, beginning at the tenth rib posteriorly, and should be carried downward below the rib margin. The absence of a kidney in that region will reveal a *muffled tympanitic sound* *Auscultation* of the kidney is valueless except for the detection of an aneurysm of the renal or adrenal arteries.

Pyelography: SEE: p 685.

The Enlarged Kidney: Enlargement of the kidneys may be caused by malignant tumors (sarcoma and carcinoma), perinephritic abscess, large multiple cyst, pyonephrosis, hydronephrosis, renal echinococcus cyst, hypernephroma and renal tuberculosis.

A mass in the right or left upper abdomen often requires a differential diagnosis between a large kidney and other conditions that may simulate it; *i. e.*, cyst, hepatic tumor, impacted colon, large spleen, ovarian cyst, suprarenal tumor, neoplasm of large intestine, omentum, mesentery or pancreas.

Inspection: A fullness of the affected side may be noted in thin individuals, particularly in the loin. A varicocele on the affected side is often present.

Palpation: The rounded poles and the "bean-shaped" outlines of the kidney is usually palpable in thin individuals; it does not descend to any great extent during inspiration, its excursion being chiefly downwards or inwards, and it may readily be pushed back into the loin

Percussion: *Anteriorly*· The large intestine usually lies in front of the kidney, therefore a tympanitic note is elicited on superficial percussion over the mass *Posteriorly*· Because of the

Differential Diagnosis, Kidney Lesions, Neuralgia and Myalgia

Symptoms	Kidney Lesions	Neuralgia (Intercostal)	Myalgia (Lumbago)
Pain.	Generally an aching dull [illegible]	Sudden sharp attacks, [illegible] ollowing an ex- [illegible] to cold or damp [illegible] mal.	Attacks are only brought on by movement, are much worse in the morning, but as the day advances, and the muscles are exercised, the pain and soreness gradually become less
Motion	Motion is generally painful, especially if of a jarring character	Motion may not increase it	Motion increases it at first, and then as the muscular activity continues the pain ceases
Stooping	Stooping, at least bending toward the side on which the lesion is located, increases the amount of pain	Stooping may not produce any change in the pain symptom	Stooping is very painful
Pressure	Pressure on the kidney through the abdomen produces severe pain Percussion is also painful Pressure points associated with the kidney zones of hyperalgesia are present.	Pressure points are present Hyperalgesic zones do not correspond to the cord zones but to the intercostal nerve zones	No pressure points but a general localized tenderness over the lumbar muscles
Muscle sensitiveness	No pain on pinching of the muscles Pinching of the skin may or may not be painful	Pressure increases the pain *Pain on pinching the skin.*	Pressure decreases the pain *Pain on pinching the muscles*
Radiation of pain	Pain has a tendency to radiate to the lower limbs, to the rest of the ilium, or to the testicles	Pain radiates around to the anterior abdominal walls	No radiation, is localized to lumbar muscles
Mobilization of the part	Strapping does not ease the aching, but may make it worse	Strapping may ease the pain.	Strapping eases the pain
Localization of pain	Patient localizes the pain *deeply.*	Patient localizes the pain *superficially.*	Patient localizes the pain *superficially*
Urine	Generally shows some sign of the kidney involvement Blood in a calculus, pus in suppuration diseases.	Urine normal	Urine normal Frequently highly acid
Fever.	Septic type in suppurative lesions Absent in non-inflammatory lesions.	Absent.	Generally absent
Pulse.	Generally increased	Normal.	Generally normal
Head zones.	May be present	Absent. Valliex's points present.	Absent. *Hyperalgesia* confined to the affected muscles

proximity of the kidney to the spinal column, dullness is elicited from the lateral aspect of the mass to the spinal vertebrae, presenting no area of resonance between the spine and the mass as is found in splenic enlargement. When a physical examination of a suspected mass fails to diagnose it definitely as an enlarged kidney, a pyelographic study should be made Pain in the lumbar region is a prominent symptom in many of the kidney diseases and it should be differentiated from intercostal neuralgia and lumbago (myalgia) The preceding table after Behan sets forth the important differential points

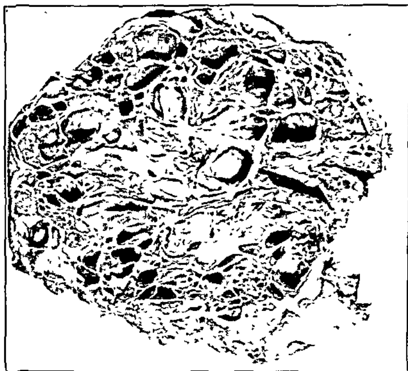


Fig 1—Cystic kidney.

Unilateral Diseases of the Kidney

The following diseases usually affect only one kidney, though in some rare instances there may be involvement of both kidneys.

Hydronephrosis

A hydronephrosis may be diagnosed by feeling a large soft fluctuating mass in the kidney region This mass may suddenly disappear only to recur the following day, or possibly several days later The disappearance of the mass when associated with polyuria indicates that the retained urine has passed through the ureter into the bladder. A more accurate diagnosis may be made by ureteral catheterization and pyelography

Pyonephrosis

A large, soft, tender, moderately-fluctuating mass, having the outline of a kidney, may be palpable in the kidney region and is associated with symptoms

of sepsis (chills, fever, sweats, and irregular temperature). Tenderness and rigidity of the muscles of the back aid in the diagnosis of this condition. The diagnosis may be confirmed by cystoscopy and ureteral catheterization, pyelography and urinalysis (the urine contains pus).

may be the seat of numerous cysts varying both in size and number. The affected kidney is usually enlarged and may be felt as a large, rounded, somewhat fluctuating, movable mass. Deep pressure over the mass may elicit characteristic *kidney sensitiveness which is* transmitted along the ureter. Polycystic



Fig 2—Hypernephroma

disease of the kidneys is often congenital and may not be discovered until the *third or fourth decade.* When most of the kidney structure is destroyed and displaced by cysts, symptoms of renal insufficiency occur, *i. e.*, hematuria, *hypertension, and progressive* anemia.

Cysts

These may be single or multiple and one or both kidneys may be affected. If sufficient uninvolved kidney structure remains to carry on their function no pathologic urinary symptoms will be manifested.

A single cyst in the kidney may be small or large, often attaining to an enormous size so that it occupies nearly half of the abdominal cavity.

Multiple Cysts of the Kidney (polycystic kidney): One or both kidneys

Perinephritic Abscess

This is often differentiated from a *large kidney because in the* former condition an induration or "bagginess" is palpable in the iliac region, while an

Differential Diagnosis of Tumors of the Kidney

Symptoms	Polycystic Kidney	Sarcoma	Hypernephroma
Pain.	Dull aching, generally in the lumbar region	Dull aching, or may be entirely painless	Generally dull aching in the back. Spasmodic, colicky pains may also occur. They are due to the passage either of blood clots or of tumor tissue through the ureter.
Urine	May show no changes until late in the disease Blood may be present	Turbid. Blood may be present.	Blood is nearly always present This is most marked when the growth has invaded the renal pelvis.
Renal colic.	Not as common as it is in other varieties of kidney tumor formation	Generally absent.	Present, time of onset varies
Tumor	Large, irregular mass in kidney region. On palpation a certain amount of resiliency is present Is often bilateral	Large, regular outline to growth.	Present, generally very large. Often the kidney can be felt on the lower pole of the mass
Age	Generally young or middle-aged adults	Generally young people.	Average between 30 and 55 years
Cachexia	None during the early stage, marked in the late stages	Present	Very common
Fever	Generally absent. When present, it indicates the beginning of suppuration	No fever	May be present

enlarged kidney can be felt anteriorly. X-ray examination with pyelography, urinalysis and cystoscopy usually aid in diagnosing and differentiating these renal conditions.

Hypernephroma

This usually occurs singly though it may produce metastasis to the other kidney, the lungs, spleen, or any other viscus. The diagnosis rests upon the finding of a large mass intimately connected with the kidney, the presence of metastasis to other organs, hematuria, cachexia, and the results of x-ray studies. Hypernephroma may originate in the kidney or the suprarenal capsule.

Amyloid Kidney

The kidney is enlarged, firm and smooth. Amyloid kidney is usually associated with amyloid disease of the liver and spleen. When the intestines are involved, diarrhea is quite common Amyloid disease of the kidney may be found in patients who are suffering from long-standing bone suppuration, *e. g.*, tuberculosis of the spine, hip, etc., or from syphilis.

Physical Examination: ***Inspection:*** The patient is pale, almost waxy in color.

Palpation: The skin is edematous, the kidney, liver and spleen are enlarged and not tender to pressure.

Urine: This contains albumin, hyaline and waxy casts, and lardacein will be found in the various tissues.

Tuberculosis

The physical examination in chronic cases *will reveal the following:*

Physical Examination: ***Inspection:*** The patient is emaciated and may or may not present a tuberculous focus in the lungs.

Palpation: The kidney region is tender to the touch A moderate degree of rigidity is felt in the lumbar muscles The urine contains albumin, pus and occasionally blood; a pyelographic study may confirm the diagnosis as will the finding of tubercle bacilli in the urine.

Carcinoma

This usually occurs in elderly people; it may be primary or secondary.

Physical Examination: ***Inspection:*** The patient is anemic, pigmentation is noted over the affected area and there is an increase in the growth of hair in both the pubic and axillary regions.

Palpation: A mass, rather soft and tender, which moves with respiration, is felt in the kidney region *Hematuria* is a constant symptom, and *cachexia* comes on early

Sarcoma

This usually occurs in the young.

Physical Examination: ***Inspection:*** A mass may be noted in the renal region, and an overgrowth of hair in the pubis and axilla.

Palpation: A large, smooth, firm, rapidly-growing mass can be felt posterior to the colon. *Very little anemia* is present but *hematuria* is a nearly constant symptom.

Floating Kidney

This is usually found in emaciated subjects or in those who have undergone

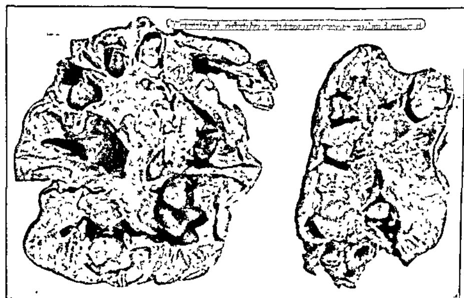


Fig. 3—Multiple renal calculi in both kidneys The right lower parathyroid was definitely hyperplastic

a severe strain. It is more common in women.

Physical Examination: *Palpation:* The palpating hand can recognize the kidney by its shape, its notch and by the fact that it can be readily moved upward to its normal position. Coughing or straining in the standing posture will again dislodge the kidney

Percussion: On percussing over the kidney region posteriorly a muffled tympanitic note will be elicited when the kidney has left its normal position.

The kidney may be slightly displaced downward by some intrathoracic condition; i e., pleural effusions or other conditions that will forcibly displace the diaphragm downward When the fat in which a kidney is imbedded is absorbed thus diminishing its proper support, it may become displaced and movable or floating The right kidney is more apt to become floating than the left kidney, because of the heavy organ (the liver) overlying it.

Renal Calculus (stone in the kidney): Renal calculi may be unilateral or bilateral The stones may be single or multiple They may be located in the pelves of the kidneys, in the calices or in other parts of the kidney. Calculus is not readily diagnosed by physical signs. Renal colic, the pain radiating downwards towards the urethra or to the inner surface of the thigh, and hematuria are characteristic symptoms; x-ray examination and pyelography are the best diagnostic means

Pyelography: A pyelographic study is indicated in cases where in addition to nephrolithiasis other pathologic conditions are suspected. Thus, the exact situation of a hard or a suspected soft stone in the ureter, pelvis or calices may be revealed. Conditions of hydronephrosis, pyonephrosis, papillomata or other growths involving the pelvis and calices, congenital and acquired abnormalities of the pelvis and ureters may all disclose themselves as the result of this study.

Pyelography may be performed by two methods: (*a*) *Intravenous pyelography*, where an opaque solution is injected in-



Fig 3a—Pyelographic study Normal pelvis

travenously (any vein in the cubital fossa) and an x-ray picture is made of the kidney regions at various intervals, and (*b*) *retrograde pyelography*, where an opaque solution is injected directly into one kidney through a catheter passed up the urethra and ureter as high as the pelvis of the kidney

(For urinalysis, see p. 967; blood chemistry, p. 1007, and kidney function tests, p. 1038 For discussion of the Adrenal Glands, see Endocrines, p. 792)

Differential Diagnosis of Intestinal, Renal, Gallstone and Uterine Colic

Symptoms	Intestinal Colic	Renal Colic	Gallstone Colic	Uterine Colic
Pain.	Generally paroxysmal, relieved at the time peristalsis produces an onward movement of gas, etc. At the time this occurs there is pronounced gurgling.	[illegible] etc.	[illegible] in the upper abdomen	[illegible] in the lower [illegible]lomen, is par[illegible]smal, and is [illegible]erally associated with a vaginal discharge of blood, frequent at time of menstrual period.
Radiation.	To upper or lower abdomen, seldom in back.	To lower abdomen and often to the testicle, or to the end of the penis on the affected side.	To the back and under the right shoulder on the right side and up to the clavicle	To thighs, external aspect and often to the back.
Urine.	No change, except that indican is frequently found.	Often a suppression for some little time and then blood is present.	Frequently bile salts and acids are present.	No change
Vomiting.	Generally present Vomitus consists of food, often undigested and fermenting. Bile may be present.	C[illegible] be present.	[illegible]	[illegible]
Tenderness.	Direct and indirect, as described under intestinal colic.	Generally over the kidney lesion and frequently the enlarged kidney can be palpated.	Generally over the gallbladder which often on palpation is found to be enlarged.	Not much present
Referred pain area.	That of intestines	That of kidney and ureter.	That of the gallbladder and ducts	That of the uterus
X-ray study.	Spastic bowel.	Pyelogram may disclose stone	Stones may be seen in the gallbladder.	No x-ray finding

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Symptomatology: The symptoms depend largely upon the severity of the infection and the kind and amount of kidney structure involved.

(*a*) **Acute Diffuse Nephritis** (hemorrhagic Bright's disease) This is characterized by an acute onset, moderately high temperature, marked edema and anasarca, rapid pulse, hypertension, delirium and vomiting The urine is scanty and high colored, contains large amounts of albumin and blood, hyaline, granular and bloody casts. Blood chemistry shows marked retention of urea nitrogen, nonprotein nitrogen, and creatinin and also some salt retention, etc

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Chronic Nephritis (Chronic Bright's Disease)

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(1) The large pale kidney, the contracted pale kidney, and the contracted dark kidney.

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Chronic nephritis like the acute variety may affect alike the entire kidney structure, or the glomerular or the tubular elements may be the principal seat of affection. The symptoms and course of the disease depend largely upon the kind and amount of tissue involved. It should be borne in mind that a sharp line of demarcation between the tubular and glomerular structures is not always observed by the pathological process, therefore, in acute and chronic nephritis one variety may eventually merge into the other, thus causing a diffuse nephritis. It is important to diagnose the variety of nephritis, chiefly because of prognosis and treatment.

Chronic Parenchymatous Nephritis (nephroses — chronic tubular or desquamative nephritis—large white kidney, chronic nephritis with edema and

Differential Diagnosis of Intestinal, Renal, Gallstone and Uterine Colic

Symptoms	Intestinal Colic	Renal Colic	Gallstone Colic	Uterine Colic
Pain.	Generally paroxysmal, relieved at the time peristalsis produces an onward movement of gas, etc. At the time this occurs there is pronounced gurgling.	Pain, paroxysmal, is found in back and is brought on by *moving, walking,* etc.	Pain is paroxysmal, generally follows an indiscretion of diet and is present in the upper abdomen	Pain in the lower abdomen, is paroxysmal, and is generally associated with a vaginal discharge of blood, frequent at time of menstrual period.
Radiation.	To upper or lower abdomen, seldom in back.	To lower abdomen and often to the *testicle, or to the* end of the penis on the affected side.	To the back and under the right shoulder on the right side and up to the clavicle.	To thighs, external aspect and often to the back.
Urine.	No change, except that indican is frequently found	Often a suppression for some little time and then blood is present.	Frequently bile salts and acids are present.	No change
Vomiting.	Generally present. Vomitus consists of *food, often* undigested and fermenting. Bile may be present.	Generally present, consists of the food most recently ingested Bile may be present.	Generally present. Vomitus is remarkably free from bile	Sometimes present, though not as frequent as in the other colics
Tenderness	Direct and indirect, as described under intestinal colic	Generally over the kidney lesion and frequently the enlarged kidney can be palpated	Generally over the gallbladder which often on palpation is found to be enlarged	Not much present
Referred pain area.	That of intestines.	That of kidney and ureter.	That of the gallbladder and ducts.	That of the uterus
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Differential Diagnosis of Chronic Parenchymatous and Chronic Interstitial Nephritis

Chronic Parenchymatous Nephritis (Tubular)	Chronic Interstitial Nephritis (Glomerular)
1 Occurs in early or middle life.	1. Occurs later in life.
2 *History of a previous acute attack, scarlet* fever, acute alcoholism	2. History of gout, lead poisoning, syphilis, excessive eating or drinking (spirits), nerve strain
3 Onset gradual or markedly manifest	3 Onset slow, insidious and indefinite.
4 Dropsy is a constant symptom	4. Dropsy is rare
5 Vascular changes and cerebral symptoms	5. Arteriosclerosis, cardiac hypertrophy and cerebral symptoms are common
6 Albuminuric retinitis common	6 Retinal hemorrhage and choking of disks
7 Marked albuminuria, many tube casts	7 Very slight albuminuria, a few casts chiefly hyaline (long, narrow)
8. *Urine* but *little* increased in quantity, often diminished, specific gravity is increased or slightly diminished	8 Urine of *very low specific gravity* and excessive quantity.
9 Anemia occurs earlier and is marked	9. Anemia slowly progressive and less marked.
10 Uremic symptoms are generally less severe, amaurosis, vomiting, diarrhea, headache	10 Uremic symptoms are generally severe, coma and convulsions, great dyspnea
11. Runs a shorter course, from two to six or seven years	11. Has a more chronic course, seven to thirty years.
12 *Blood chemistry—salt retention*	12 *Blood chemistry, nitrogenous products* retention

salt retention—degenerative nephritis). The onset is gradual, becoming progressively worse

Etiology: It may follow the acute variety; and infections, fevers, septicemia *and alcohol are among the predisposing* factors

Symptomatology and Diagnosis: The patient is weak and suffers from indigestion. The skin is pale, the pallor is greater and out of proportion to the blood count. Edema of the lower eyelids and later general edema is present; while in severe cases anasarca with effusion in the serous sacs, dyspnea and albuminuric retinitis may be noted. When the glomerular structures become involved, the blood pressure rises and the quantity of urine diminishes. Uremic symptoms may manifest themselves, *i. e.*, headache, vertigo, sleeplessness, nausea, vomiting, stupor, convulsions and coma. The urinous odor on the breath should not be mistaken for the urinous odor about the patient when incontinence of *urine exists.*

Urinalysis and Blood Chemistry (for details see pages 967 and 1007). *The urine is scanty, of high specific* gravity and contains large quantities of albumin, and tube casts (epithelial, red corpuscles, *and hyaline and granular*). The blood shows retention of sodium chloride. When the glomeruli are affected, *nitrogen retention is noted.* Kidney function tests reveal the inability of the kidney to excrete salt and water. The dye tests (*phenolphthalein or indigo* carmine), show great retention (See: *Renal Function Tests,* p. 1038).

Chronic Interstitial Nephritis (hemorrhagic nephritis, chronic glomerular nephritis, contracted kidney, chronic nephritis without edema and with hypertension and nitrogen retention in the blood) In this subvariety of chronic nephritis, the glomerular elements of the kidney structure are principally involved.

Etiology: It may be superimposed upon or it may follow chronic parenchymatous (tubular) nephritis Alcohol, lead, syphilis, irritating toxins and bacterial invasion are among the etiologic factors.

Symptomatology and Diagnosis: The common symptoms are digestive disturbances, headache, weakness, disturbance of eyesight with retinal hemorrhages The skin is usually dry and only slight edema of the ankles may be present Tingling in the fingers with blanching and other vasomotor disturbances are often found. Hypertension is marked The urine may contain blood; it is of low *fixed* specific gravity, and the night output may equal that of the day output Albumin is usually scant (reported as a trace) Tube casts are few, of the narrow hyaline type, sometimes granular and bloody casts are found The blood shows great retention of urea, uric acid, nonprotein nitrogen and creatinin. Uremia is a frequent complication Kidney function tests show poor concentration The urea clearance is low.

Arteriosclerotic and Senile Kidney (nephrosclerosis, vascular nephritis): Essentially the arteriosclerotic kidney, the senile kidney, and chronic interstitial nephritis of other writers present similar manifestations, excepting that the arteriosclerotic and senile kidney conditions are usually found in persons who have primarily developed arteriosclerosis or become senile. The kidneys like most of the organs in the body have participated in the sclerotic change, therefore hypertension, polyuria, etc., are found, while primary chronic glomerular or interstitial nephritis is the initial disease which produces sclerotic changes even in the young

Symptomatology and Diagnosis: Usually this condition attacks persons over 50 years of age; it is characterized by progressive weakness and inability to withstand physical or mental strain The skin is dry, often covered by scales or eczematous eruptions Tinnitus, vertigo, polyuria, nocturia, hypertension, sclerotic corneal vessels, liability to cerebral hemorrhage, dyspnea and myocardial changes occur frequently. The urine is large in quantity, of low specific gravity, contains little albumin and few small, narrow hyaline casts

Blood: The blood presents a picture of secondary anemia, and the blood chemistry reveals nitrogen retention, e g, increased amounts of urea, nonprotein nitrogen and creatinin

Uremic Coma

This condition occurs as a result of disturbed kidney metabolism and is found in the presence of nephritis as a result of insufficient elimination from the blood of certain toxic substances normally excreted by the kidneys

Inspection: The patient is stuporous and respiration stertorous. No change in pupillary reaction is noticeable Convulsions, twitchings and coma are common

Palpation: The skin is dry, the pulse hard and rapid, and the blood pressure is elevated. There is generally a urinous odor on the breath; this, however, should not be confused with the urin-

Differential Diagnosis of Coma in Uremia, Cerebral Hemorrhage and Alcoholic Narcosis

Uremia	Cerebral Hemorrhage	Alcoholic Narcosis
Pupils generally dilated; *albuminuric retinitis.*	Pupils unequal or dilated.	Pupils contracted or dilated; eyes injected.
Sharp, hissing stertor	Stertorous, puffy breathing, and flapping cheek	No stertorous breathing
Urinous odor.	*No odor.*	Odor of alcohol.
No paralysis.	Paralysis; hemiplegia.	No paralysis, usually
May or may not be aroused	Unconsciousness absolute	May be aroused.
Pulse at first strong, later weak and rapid; tension hard, arteriosclerosis.	Pulse slow and strong or irregular; arteries often atheromatous	Pulse frequent and feeble
Coma gradual or sudden	Coma sudden and deep	Coma gradual
Preceded by general convulsions, headache, etc.	Convulsions late; may be unilateral.	No convulsions.
Urine albuminous.	Urine generally negative	Urine generally negative
Edema and pallor; heart hypertrophied.	Heart may show hypertrophy	Red face and nose, heart often weak, myocarditic.

ous odor about a patient suffering from incontinence. The urine is scanty and contains albumin and many casts; at times there is complete retention of urine; the blood shows retention of nitrogen, urea and creatinin.

Chronic Uremia: This is characterized by headache, dizziness, anorexia, vomiting, feeble heart action, visual disturbance, scanty urine and retention of nitrogenous products in the blood.

Congestive Kidney

Passive congestion of the kidneys occurs as the result of myocarditis during the stage of decompensation.

Symptomatology and Diagnosis: The patient is cyanotic, dyspnea is marked, the heart is dilated and shows other evidence of decompensation. The lungs are edematous, anasarca is well marked, with the greatest amount of edema in the dependent parts of the body. The urine is scanty, dark and of high specific gravity, containing much albumin and only a few hyaline casts. The blood chemistry shows hypoproteinemia and very little retention of nitrogenous products. This condition is relieved when cardiac compensation is restored.

Pyelonephritis and Other Infections of the Kidneys

Pyelonephritis: This results from the invasion of the kidney by pathogenic organisms through various routes. These infections may occur retrograde from the lower urinary tract or genitalia; by direct extension from other organs; by way of the blood stream and through the lymphatics. The infection may be acute or chronic, bilateral or unilateral.

The symptoms depend upon the type of infection and the extent of renal damage. These are usually chills, irregular type of septic temperature, headache and malaise. The urine may contain albumin and pus in varying amounts and bacteria may be found on culture. The urine is acid in colon bacillus infection and alkaline in Proteus Vulgarus infection. In the chronic type the symptoms of infection are milder than in the acute type,

but there is evidence of a greater degree of kidney destruction. This may give signs of severe glomerular, parenchymatous or diffuse nephritis *plus* pyuria and bacilluria, and it may terminate in uremia.

Pyelitis: This is an infection of the *pelvis of the kidney. It may occur as an* ascending infection or be caused by obstruction to the *outflow of urine from* the kidney. This is seen fairly often in baby girls and in pregnant women, *it* may result from a twisted ureter or from obstruction by stone, tumor or other conditions that interfere with free drainage. The symptoms are fever, chills, burning and frequency of urination, pyuria and tenderness on palpation over the affected flank. Urethral catheterization and pyelography will determine the site of infection, and urine culture the type of infection.

Toxemic Kidney (Toxic Nephrosis)

Degenerative rather than inflammatory lesions in the kidney may be caused by certain endogenous and exogenous substances which affect chiefly the convoluted tubules causing various degrees of parenchymatous degeneration

The endogenous causes of the so-called febrile albuminuria are acute specific fevers such as pneumonia, typhoid fever, smallpox, diphtheria, etc Tonsillitis, scarlet fever, *toxemia of pregnancy*, jaundice, diabetes mellitus, and other toxic substances *in the blood may* cause toxic nephrosis, but are likely eventually to cause a true *nephritis*.

The exogenous causes are various metallic *poisons such as mercury*, bismuth, arsenic, phosphorus, etc., and nonmetallic substances *such as cantharides* and other renal irritants The urinary findings are albuminuria, tube casts, leukocytes and rarely a few erythrocytes. The blood shows no evidence of nitrogen retention.

Symptoms: There may be various degrees of edema, some headache, occasionally *dimness of vision*. The eyegrounds may occasionally show some edema *of the discs or partial detachment* of the retina; the vessels appear normal; *hemorrhages are rare.*

Lipoid Nephrosis

This is a degenerative process, as pointed out by Epstein, affecting the epithelium of the convoluted tubules. It is questionable *if nephrosis is a true* renal inflammatory disease. The manifestations are those of disturbed endocrine and cholesterol metabolism affecting the renal tubules.

Symptomatology and Diagnosis: The most characteristic symptoms of this condition are well-marked edema or anasarca, low blood pressure, moderate and progressive anemia and low basal metabolism. The urine contains a large amount of albumin, many casts but no erythrocytes

Blood Chemistry will show great salt retention, normal urea, nonprotein nitrogen and creatinin; decrease in the total blood protein with an increase of globulin and a great increase in the cholesterol. The retinal vessels are *normal* Nephrosis *may at times merge* into nephritis.

Whether lipoid or Epstein's nephrosis is a renal disease entity or only a local *manifestation of a general systemic* disturbance is a mooted question. It would *appear that the edema has little relation* to the kidneys, but that it depends on an *altered state of capillary permeability*, the cause of which is unknown. It may be *toxic or nutritional* Kaufmann and Ma-

son believe that nephrosis as applied to the kidneys is an early manifestation of a general systemic cellular degenerative process of unknown origin The lowered basal metabolism must be classified as a secondary hypothyroidism. The thyroids of these patients can manufacture thyroxin at a normal rate, but, due to the lack of tissue call, the thyroxin con tent of the tissues falls below normal This results in altered cellular nutrition The true nephrotic kidney progresses into the secondary contracted type as a result of an organizing process of the degenerated cells, *and not as a primary* inflammatory entity. The pathologic findings, in the different types, vary according to the stage in the disease at which death occurs

Nephrosis if it persists will gradually develop into nephritis, the so-called nephritic stage of nephrosis. Occasionally *nephritis may develop signs of nephrosis*, the so-called nephrotic stage of nephritis

The Bladder

Physical Examination of the Bladder

The urinary bladder is situated in the lowermost portion of the pelvis and lies below the symphysis pubis. The empty bladder cannot be detected by physical examination, but when greatly distended it can be felt as a fluctuating globular mass in the lower midabdomen When paralysis of the bladder or great retention of urine occurs, the bladder may become enormously distended, reaching halfway up to the umbilicus Vesical calculus, carcinoma, papilloma, tuberculosis and foreign bodies, may cause hematuria The diagnosis of these conditions is best made by the use of the cystoscope and *x*-rays

Diseases of the Bladder

The urinary bladder unlike most of the other organs of the body has no function other than that of a receptacle. It receives the urine secreted by the kidneys, which is brought to it by the ureters and is expelled from the body through the urethra

Disease of the bladder therefore gives rise to no systemic manifestations, unless the disease is a systemic one, *i e.*, carcinoma, tuberculosis, etc. On the other hand there are quite a number of conditions that may so irritate the bladder as to cause local inflammation of its mucosa, known as cystitis

Cystitis

By this term is meant an inflammation of the inner lining of the bladder This condition may be caused by a variety of factors, *i e., traumatic, mechanical, chemical* and *biological.*

Traumatic Causes: Cystitis due to trauma of the bladder wall may result from violence such as fracture of the pelvis, causing rupture of the bladder, stab wounds or gunshot wounds perforating the bladder, injury to the bladder during childbirth, and the clumsy insertion of a catheter in the male urethra through a false passage. These cause bladder irritation because of injury to the bladder wall

Mechanical Causes: Here may be mentioned the presence of foreign bodies in the bladder such as pins, hairpins, *wood splinters, catheters* (either allowed to remain too long in the bladder as a retention catheter, or when one has *accidentally slipped back*), *stone, tumors,* instrumental injury by catheter or cystoscope and various parasites, such as *roundworms or pinworms.*

These cause cystitis because of direct injury to the mucosa of the bladder.

Chemical Causes: These are of two kinds: First, in which a strong chemical substance, such as a strong potassium permanganate solution, a strong silver solution, or any other irritating chemical substance has been introduced into the bladder by the urethral route, and second, in which a highly irritating substance is brought to the bladder by way of the kidneys as in poisoning by bichloride of mercury, phenol, oxalic acid, etc, or by the prolonged administration of large doses of sandalwood oil, turpentine, copaiba, cantharides and alcohol

Biological Causes: This group embraces the commonest causes of inflammation of the bladder The infection may be brought to the bladder by way of the urethra, the ureters, the kidneys, the adjacent structures and by the circulation The offending organism may be the colon bacilli, tubercle bacilli, strepto- and staphylococci or any other microorganism that may attack a previously inflamed or injured bladder or a perfectly normal bladder

Symptomatology: Cystitis, no matter of what origin, presents the following symptoms· Frequent urination, often painful and associated with tenesmus or a sense of heaviness and discomfort in the bladder region In some cases retention of urine is a troublesome feature The urine is usually cloudy, of alkaline reaction and has an ammoniacal odor. Microscopically the urine contains bladder cells, often pus and blood.

Physical Examination: A distended bladder may be palpated above the symphysis pubis, but when the bladder is empty, it cannot be palpated. Bladder tenderness may often be elicited by palpating the bladder per rectum or vaginally A cystoscopic examination and urinalysis are the best means at our command for the detection of cystitis.

Vesical Calculus

A stone in the bladder may be of kidney origin, that is, a renal calculus may be passed down into the bladder, it may remain there for some time, without increasing in size, or it may gradually become larger because of the addition to its bulk of uric acid or other substances. The presence of a stone in the bladder from any source, because of irritation, may produce congestion and at times infection and inflammation, thereby causing cystitis A characteristic symptom of vesical calculus is the sudden stopping of the stream during urination in the erect posture. Tenesmus, frequency of urination, and at times also dribbling may occur. The urine is usually that of a cystitis with or without hematuria The urethral sound, the cystoscope and the x-rays are the best means for diagnosing this condition



Fig 4—Papilloma of the bladder.

Tumors of the Bladder

These may be sarcoma, carcinoma, papilloma or any of the benign forms

Symptomatology: A small tumor in the bladder which does not bleed, may entirely escape detection. When the tumor becomes large, it may cause vesical tenesmus, a sense of weight in the bladder, frequent urination and other signs of cystitis. Malignant tumors, particularly papillomata, bleed early in their course. Therefore the presence of blood in the urine should always be investigated by a cystoscopic examination.

Tuberculosis of the Bladder

Tuberculosis of the bladder may be secondary to a tuberculous kidney, generalized tuberculosis or, in rare instances, it may occur as a primary disease of the bladder.

Symptomatology: The distribution of the ulcers, their number and probably their size determine the urgency of the symptoms. When an ulcer occurs over the vesical sphincter it will give rise to great frequency of urination with distress. The general symptoms of tuberculosis of the bladder are those of severe cystitis with frequent bleeding. The presence of cystitis in a tuberculous individual should arouse suspicion of vesical tuberculosis. A cystoscopic examination, and a careful microscopic examination of the urine may reveal the cause of the infection. When in doubt a guinea pig may be inoculated with a few cubic centimeters of a centrifuged fresh specimen of urine.

Irritable Bladder

In addition to the conditions that may cause bladder irritation and cystitis already described, it is well to mention *enlarged prostate* in the male and *retrodisplaced uterus, pelvic tumors* and *prolapsed uterus* in the female. These conditions, because of pressure upon the bladder or its outlet, may cause urinary retention with subsequent infection resulting in cystitis and, at times, in hematuria.

Irritable bladder manifested by frequent urination may at times be a *nervous manifestation* This is often seen during periods of stress and excitement. In these cases the frequency is diurnal.

Diverticulum of the Bladder

This is a local ballooning out of a portion of the bladder; it may be single or multiple. It is usually due to loss of elasticity of a portion of the bladder wall. There often is a considerable retention of urine in the diverticulum which may cause cystitis. When there is much retention it may be palpable as a tumor mass above the symphysis pubis The diagnosis of diverticulum is made by cystoscopy and cystography.

The Genitalia

Examination of Female Genital Organs

The female generative organs are examined by inspection and palpation. The pregnant uterus after the sixth month of gestation is also examined by auscultation in order to detect fetal heart sounds and the uterine souffle.

Inspection: The external genitalia is inspected as to the size of the labia and clitoris, the presence of edema, rashes such as boils, eczema, syphilitic rashes, granulomata and elephantiasis The vulva is examined for discharges, rashes and signs of inflammation. The urinary meatus is examined for inflam-

mation, discharges, polyps, carcinoma and caruncle.

The perineum and vaginal vault are examined for signs of inflammation, tears, rectocele and cystocele. The vagina is inspected through a speculum, the condition of the walls and the presence of secretions are noted The uterine cervix is likewise inspected through a speculum and the following should be noted:

The condition of the cervix, whether large or small, intact or lacerated; the presence of discharge, its consistency, quantity and odor (a specimen may be taken on a platinum loop for microscopic examination); ulcerations of the cervix, denuded mucous membranes and cysts if present should be thoroughly inspected Prolapses of the uterus and degree of prolapse, as well as the presence of hernias, are to be noted.

Palpation: The gloved hand is lubricated and the index and middle fingers are gently inserted into the vagina, the patient assuming a dorsal flexed position. The strength of the perineum is tested The cervix is palpated as to hardness, degree of mobility and tenderness The fundus uteri is palpated bimanually, one hand is placed over the lower abdomen and with the fingers of the other hand in the vagina the fundus is located, its size is thus noted, also its degree of mobility and its position. Douglas' pouch is then palpated for the presence of a mass, fluctuation or inflammatory exudate

The ovaries when normal are not easily palpable but when inflamed or enlarged they may be detected by palpation The fallopian tubes are usually unpalpable when normal; an inflamed tube or a pyosalpinx (pus in the tube), may be detected by its size and doughy feel. Differentiation is at times necessary between a distended bladder, ascites, ovarian cyst, dermoid cyst, pregnancy, uterine fibroid, myoma or other uterine tumors.

Diseases of the Female Genital Organs

Diseases of the Vulva

In considering the diseases of the vulva, affections of the following structures are to be included: The lower portion of the mons veneris; the labia majora; the labia minora; the clitoris; the hymen; the urinary meatus, and Bartholin's glands

Inflammations of the Vulva: The skin covering the vulva may be the seat of various ***skin lesions*** such as dermatitis, eczema, herpes, erysipelas, dermaphytosis, or other types of skin irritation which may cause itching, burning or pain

Gonorrhea: This may affect the vulvae of children but seldom of adults, because of the protection afforded by the many-layered mucosa of the adult vulva The vulvar gonorrheal infections of adults is limited to the vulvovaginal glands, the urethra and Skene's ducts. Gonorrheal urethritis and infection of Skene's ducts are recognized by inflammation and tenderness of the part and by a purulent discharge which contains the gonococci

Bartholinitis: This is an infection of the vulvovaginal glands and is, in the majority of cases, due to gonococcal infection. The acute stage is characterized by swelling, edema, engorgement and pain of the gland and its adjacent structure, and the affected gland usually contains pus or becomes abscessed. Chronic bartholinitis is characterized by enlargement and induration of the gland.

Symptomatology: A small tumor in the bladder which does not bleed, may entirely escape detection. When the tumor becomes large, it may cause vesical tenesmus, a sense of weight in the bladder, frequent urination and other *signs of cystitis* *Malignant tumors*, particularly papillomata, bleed early in their course. Therefore the presence of blood in the urine should always be investigated by a cystoscopic examination.

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Ulcerative Lesions of the Vulva: Simple ulcers, single or multiple, may affect the vulva or lower portion of the vagina; they may be due to nonspecific irritation or to the Bacillus crassus, which is often a normal inhabitant of the vagina.



Fig 5—Granuloma inguinale.

Chancroid: This forms a ragged irregular ulcer, it is not indurated though it appears excavated and has a granulating and often purulent surface. It may cause edema of the adjacent structures The causative agent is the bacillus of Ducrey, which may be found in the exudate.

Granuloma Inguinale: This is a specific venereal disease nearly always found in the negro, characterized by the formation of superficial ulceration covered with granulation tissue usually affecting the labia minora, the mons veneris and may spread over the entire vulva, the pubic and the inguinal regions The specific cause is said to be the Donovan bodies (SEE: Fig. 5).

Lymphogranuloma Inguinale (*Lymphopathia Venerium*): This begins as a small lesion upon the genitals and is followed within 10 or 20 days by a slowly developing unilateral inguinal adenitis. As the disease progresses, there may develop extensive ulceration with productive inflammation which may result in large tumorlike elephantiasis masses (SEE: Fig. 6) or in extensive ulceration involving the labia, the perineum, the anus and lower rectum. The inguinal adenitis is progressive and may attain a large size, being painful and suppurative. The ulcerative lesions are known as esthiomene.

This disease is of venereal origin; it is seen chiefly in the negro race, but occurs also among white males and females. The specific cause is attributed to filtrable virus. The Frei test usually becomes positive within 10 to 20 days after exposure and remains positive throughout life.

Syphilitic Lesions of the Vulva: These may be primary, secondary or tertiary lesions.

Chancre: This is the primary lesion of syphilis of the vulva It is a firm nodular lesion with slight superficial ulceration and a moderate amount of induration (less induration than in the male) or it may occur as a punched-out ulcer, having a hard base, which is indurated, clean and painless, and may be single or multiple, affecting usually the labia majora and minora and often nearby structures.

Condylomata Lata: These are the secondary lesions of syphilis of the vulva They are flattened, moist papules (wartlike structures) raised only slightly above the surrounding tissue, having a grayish necrotic appearance with a somewhat depressed center. These

lesions may affect the vulva, the perineum, the perianal region and the inner surface of the thighs. Occasionally these warts may coalesce and form large ulcerative masses having a foul discharge

Tertiary Lesions of Syphilis: These are either gummata or ulcers which may destroy the vulva and adjacent structures



Fig 6—Lymphogranuloma inguinale elephantiasis of vulva.

Diagnosis: The diagnosis of the primary lesion may be confirmed by the finding of the spirochete pallida The secondary and tertiary lesions also contain the spirochete and the patient's blood yields a positive Wassermann, Kahn, Kline or other serologic test for syphilis

Tuberculosis of the Vulva: This is an uncommon lesion. It begins as a nodule which later ulcerates and appears as an irregular punched-out ulcer with undermined edges, grayish in appearance, having a purulent or caseating exudate. The diagnosis may be made by the finding of the tubercle bacilli in the pus, by biopsy showing the characteristic tubercle formation, or by the result of guinea pig inoculation.

Kraurosis of the Vulva: This is characterized by atrophic changes in all the structures of the vulva. The tissues are atrophic, thin and appear brittle or glistening. It occurs frequently in old women or during the menopause. Pruritis is a troublesome symptom in this condition

Leukoplakia of the Vulva: This is characterized by the occurrence of white patches, either isolated or generalized, over the labia and perineum. It is associated with atrophic and sclerotic changes and, in most cases, causes severe itching, vaginismus, and often inflammatory changes

Tumors of the Vulva: These may be benign or malignant.

The Benign Tumors: These may be cysts (of the Bartho'in glands or wolffian duct), and solid tumors such as papilloma, lipoma, hydradenoma of the sweat glands (syringocystadenoma), condyloma acuminatum, fibroma, fibromyoma, urethral caruncle, angioma and the various granulomata

Malignant Tumors: These may be primary or metastatic, they are carcinoma, sarcoma, melanoma, and teratoma.

Carcinoma is the commonest of the malignant tumors It may arise from the labia majora or minora, the clitoris, the vestibule, from Bartholin's gland and from the urethra, or it may be secondary to carcinoma elsewhere. The initial lesion may be a small nodule which has

casionally, however, there may be more severe hemorrhage.

Tuberculosis of the Cervix: This is a rare condition and is characterized by ulcerations which have a peculiar grayish appearance and are covered with cheesy exudate. Tubercle bacilli may be found in the exudate and a biopsy may show characteristic tubercle formation

Cysts of the Cervix: The cervix may undergo cystic degeneration so that a great portion of it is affected, or the cervical gland ducts may become obstructed, forming nabothian cysts or follicles arranging themselves as a ring around the cervical os This is easily recognized by inspection The cervix is thick and the cysts are easily recognized

Malignant Disease of the Cervix: ***Carcinoma:*** This is a fairly frequent disease and it may be of two types · (1) The epidermoid or squamous carcinoma which arises from the stratified squamous epithelium, and (2) the adenocarcinoma which arises from the cylindrical gland-forming epithelium.

The location of carcinoma may be either extracervical or endocervical

Diagnosis· Diagnosis of carcinoma during the very early stage is sometimes difficult. The cervix presents a somewhat elevated superficial layer of tissue which has a somewhat wrinkled surface; later the growth becomes larger, has a fairly hard base and bleeds easily. The symptom most suspicious of carcinoma of the uterus (cervix or fundus) in a woman past her menopause is hemorrhage Occasionally unexplained hemorrhages may occur during the menopause; however, unusually prolonged bleeding or frequent small hemorrhages, or bleeding after sexual intercourse should always be thoroughly investigated for possible malignancy. Any suspicious lesion of the cervix or a history of bleeding in a woman of the cancer age requires exploration of the interior of the uterus by curettage. In the presence of a visible lesion of the external os a biopsy is to be taken, and the scrapings as well as the tissue obtained by biopsy should be examined microscopically.

Nonmalignant Causes of Uterine Bleeding: These may be: Cervical polyps; cervical erosions due either to syphilis or to tuberculosis or to other causes; congestion of the uterus due to malposition; retention of clots; endometriosis; inflammatory strictures of the cervix; nonmalignant uterine and ovarian neoplasms; aplastic anemia; acute leukemia; purpura; supersaturation with estrogenic hormone; lack of lutein hormone; vitamin K deficiency, and mitral stenosis.

Diseases of the Uterine Body (Fundus Uteri)

Among the commoner diseases of the fundus of the uterus are endometritis, cysts, myometritis, benign or malignant tumors and polyps.

Endometritis (inflammation of the inner lining of the uterus).

In health the endometrium shows various changes during the menstrual cycle and during pregnancy. In disease, the endometrium may be the seat of acute or chronic inflammation.

Acute Endometritis: This is the result of some acute inflammatory process affecting the endometrium; it may be secondary to pelvic infection, to extension of infection from the vagina through the cervix as in gonorrhea, or it may be introduced by instrumentation. Acute endometritis may also result from puerperal sepsis, retained secunda or blood clots. The inflammation may be super-

ficial or it may extend to the myometrium and it may cause suppuration. In acute infection there is fever, tenderness over the uterus and its adnexa, and a foul-smelling discharge. Specific endometritis is of gonorrheal origin.

Chronic Endometritis: This condition is quite common; it may follow acute endometritis or it may be due to chronic infection or to chronic disease of the cervix, tubes or ovaries, or to uterine displacements. The *symptoms* are, frequent bleeding, considerable uterine discharge and often menstrual disturbances such as menorrhagia, metrorrhagia or dysmenorrhea.

Senile Endometritis: This is a form of chronic endometritis which may cause postmenstrual bleeding. This condition is to be differentiated from adenocarcinoma.

Tuberculosis of the Endometrium: This is generally secondary to tuberculosis of the tubes, the ovaries or the lower genital tract; occasionally no primary focus is found elsewhere.

Diagnosis: The diagnosis of the various types of endometritis can only be made by histologic examination of the endometrium after curettage, and by bacteriologic examination of the uterine discharge.

Myometritis: Disease of the uterine muscle may be acute or chronic. Acute myometritis is usually associated with acute endometritis and is found in various septic conditions Chronic myometritis may be associated with chronic endometritis resulting from gonorrhea or other infection that has either gone through an acute stage or started as a mild chronic invasion. In both the acute and chronic types of myometritis the uterus may be enlarged; it is, however, more tender in the acute stage and is harder in the chronic stage.

Endometrial Polyps: Polyps of the endometrium may be divided into three types· (1) Those made up of functional endometrium; (2) those of immature endometrium, and (3) those composed of endometrial elements and voluntary muscle tissue. Uterine polyps, irrespective of their structure, may cause uterine bleeding. Microscopic examination of the polyp will usually reveal its histologic structure.

Cysts of the Uterine Cavity: These are rare. They may be congenital or they may follow puerperal or other infections, or they may be caused by cystic degeneration or necrosis of a myoma.

Benign Tumors of the Uterus: The commoner tumors of the uterus are myoma and adenomyoma.

Myoma: Myoma of the uterus, often spoken of as fibroids, is exceedingly common; it may occur in the young or old and is generally noted in the third decade. The growth may be subserous and pedunculated, or it may be intramural (interstitial). These tumors may be single or multiple, and may vary in size from that of a walnut to that of a watermelon The submucous variety usually impinge upon the blood vessels of the endometrium and cause free bleeding. *As the tumors continue to grow* they invade the uterine cavity and cause distortion and enlargement of the cavity of the uterus The interstitial myoma, when small, may cause no change in the contour of the uterus; and when they attain larger sizes they cause enlargement with some irregularity in the contour of the uterus They cause bleeding less frequently than do the submucous variety. These tumors may arise from the fundus or from the cervix The diag-

nosis of a uterine growth is easily made by palpation. Its exact type, however, is more definitely diagnosed after operation and microscopic examination of the removed tissue. Myoma may undergo various changes such as hyaline or cystic degeneration, calcification, necrosis, infection, fatty changes and malignant changes.

Adenomyosis or Adenomyoma: This does not cause a definite circumscribed growth but a rather generalized infiltration of the uterine muscle. It is seldom very large. The posterior wall of the uterus is usually larger and thicker than is the anterior, though occasionally the entire uterine muscle is thickened The uterus is fixed, and is not tender to palpation Adenomyosis is often found as a result of chronic pelvic inflammatory disease and only occasionally may it be associated with distinct myoma of the uterus.

Malignant Tumors of the Uterus: These are carcinoma, chorionepithelioma, sarcoma, hydatiform mole, placental rests and polyps.

Carcinoma of the uterus is the commonest malignant tumor of the uterine fundus; it usually occurs in women past the age of fifty, though it may occur at an earlier age The type of carcinoma is usually adenocarcinoma, malignant adenoma and squamous cell carcinoma.

Adenocarcinoma: The tumor may affect the entire uterine cavity and may descend into the cervix. The two prominent symptoms are some enlargement of the uterus and metrorrhagia. The bleeding may be moderate or profuse and may occur at irregular intervals When the mass undergoes necrotic change, there is a foul vaginal discharge.

Malignant Adenoma: This usually occurs as a papillary luxuriant endometrial growth; it infiltrates the uterine wall, causing an asymmetrical, soft enlargement. This type of tumor also causes bleeding. The diagnosis is made from the examination of the uterine scrapings.

Squamous Cell Carcinoma: This is rather rare. It may occur either as a distinct entity or in association with other malignant types The uterus usually enlarges and, as in other types of carcinoma, early bleeding or profuse discharge is a prominent symptom

Sarcoma of the Uterus: Any portion of the uterus may be invaded by this type of tumor though the body is more frequently involved than is the cervix The uterus may become somewhat enlarged, other symptoms are bleeding and discharge, though both may be absent. Metastasis occurs early by direct continuity, by the blood stream or by the lymphatic stream From the clinical point of view it is not possible to differentiate between carcinoma and sarcoma unless there be hematogenous metastasis

Chorionepithelioma: This is a tumor of the embryonic chorion, it may develop after an abortion or during pregnancy The growth springs from the chorionic villi and invades the uterine wall, the blood channels and the uterine musculature with trophoblastic cells causing destruction of uterine tissue and hemorrhage. Occasionally this tumor may develop beneath the surface, within the uterine wall The clinical findings are enlargement of the uterus, uncontrollable uterine hemorrhage, and a positive pregnancy test though the fetus be dead or absent. The *diagnosis* is definitely made by microscopic examination of the tissue obtained by uterine curettage. This tumor is of rapid growth and may

cause early hematogenous metastasis in the vagina, lungs, brain, liver, kidneys and other structures.

Malignant Hydatiform Mole: This is a rounded mass containing clusters of grapelike vesicles It may be small, having few vesicles, or large and containing many. This tumor also develops from the chorionic villi; it is usually found in association with some product of pregnancy. The uterus usually enlarges out of proportion to the length of pregnancy. There is uterine bleeding during the early months of pregnancy. Pregnancy tests are generally positive. Hydatiform moles are considered by some authorities as being akin to chorionepitheliomas

Placental Rests and Polyps: These may remain dormant in the uterus for a considerable time and undergo malignant change during pregnancy or because of acute or chronic inflammation of the uterus The chief *symptoms* are profuse and persistent bleeding during pregnancy and bleeding with subinvolution of the uterus after completion of pregnancy Curettage and examination of the scrapings usually disclose the diagnosis

Disease of the Fallopian Tubes

Diseases of the Fallopian tubes include salpingitis, tuberculosis, tubal pregnancies and tumors

Salpingitis: This term denotes inflammation of the tubes, one or both tubes may be affected The inflammation may extend to the ovaries or uterus, and may be acute or chronic. Acute salpingitis may be caused by gonococci, staphylococci, streptococci, colon bacilli, or tubercle bacilli.

Gonorrheal Salpingitis: This is the most frequent type encountered; it is secondary to vaginal or cervical gonorrhea. The infection usually causes an endosalpingitis which spreads to the other layers of the tube, causing either partial or complete tubal occlusion with suppuration and enlargement. The chief *symptoms* are pain, tenderness, and septic temperature. On *examination,* the tube may be felt as a large, round, tender mass and there may be an associated cellulitis, or a pelvic abscess in the tubo-ovarian region. The disease may be unilateral or bilateral.

Pyogenic Salpingitis: This may follow abortion, surgical operation on the cervix, uterine curettage, or it may be caused by other types of infection. The *symptoms* are severe pain in the pelvis, septic type of temperature, tenderness in the region of the broad ligament with cellulitis, phlebitis, lymphangitis, and, at times, abscess of the broad ligament.

Chronic Salpingitis: This may be manifested as pyosalpinx, hydrosalpinx or chronic interstitial salpingitis.

Pyosalpinx (pus tubes): This is usually the result of gonorrheal salpingitis, though it may also occur in tuberculosis or pyogenic infection There is usually a blockage of the lumen of the tube at the fimbriated end which may cause occlusion of the entire tube. Examination will reveal an enlarged tube, some chronic pelvic inflammatory manifestations, and a purulent discharge.

Chronic Interstitial Salpingitis This is characterized by enlargement of the tube and thickening of its wall. The enlargement may be moderate or pronounced, depending on the volume of the tube content and the thickness of its wall. The *symptoms* are pain or fullness in the pelvic region, often accompanied by a nonpurulent cervical discharge. Pelvic *examination* will reveal

tenderness and enlargement of one or both tubes.

Hydrosalpinx: This may result from pyosalpinx or from other inflammations causing tubal occlusion. It is usually an exceedingly chronic condition and may tend to form a tuboövarian cyst. On *examination* a cystlike mass, either cylindrical or rounded, of varying size, may be found in the affected tuboovarian region.

Tuberculous Salpingitis: This is fairly common, and according to Novak,[1] comprises about 5 per cent of all cases of salpingitis. The tubercle bacillus may reach the tubes by the hematogenous route or the infection may spread to the tubes in the genital tract. When it occurs as a primary disease of the tubes it may spread to the cervix and to the vulva. The *symptoms* are irregular fever of low degree, pain and tenderness in the tubal region, leukorrheal discharge, and, when the vulva is infected, characteristic ulcerations are noted. Tubercle bacilli may be found in the infected tissue or in the discharge.

Tubal Pregnancy (ectopic pregnancy): The cause of tubal pregnancy is not entirely known Often tubal pregnancy remains unrecognized until the tube ruptures and severe hemorrhage results. The history of a missed period with sudden pain in the iliac region and the occurrence of slight or moderate vaginal bleeding often causing shock, and the finding of a mass in the tubal region should call attention to the possibility of a ruptured tubal pregnancy.

Tumors of the Fallopian Tubes: These may be malignant or benign.

[1] Novak, Emil: Gynecology and Obstetrical Pathology, p 223, W. B Saunders Co, Philadelphia, 1940

Malignant Tumors: ***Carcinoma:*** This may be primary or it may be secondary or metastatic from the uterus or other pelvic structures. Carcinoma is generally found during the middle period of life. The *diagnosis* may be suspected by finding a hard mass in the tubal region that may cause a moderate amount of pain, bleeding, and some discharge.

Other Types of Malignant Tumors These are chorionepithelioma, adenomyoma and sarcoma.

Benign Tumors: These are fibroma, fibromyoma and cysts. They have no definite distinctive clinical characteristics. On *examination* a mass may be discovered in the tubal region which may be fixed and somewhat tender. Various tumors may also occur in the round ligaments, in the broad ligaments and in the intrasacral ligament.

Disease of the Ovaries

The ovaries have a double function due to their internal and external secretions. Disease of the ovaries may, therefore, cause definite endocrinopathies such as disturbance in menstruation, sterility, disturbance in somatic and sexual development; and it may also cause other nonendocrine defects because of inflammation, tumors and other pathologic change of their structure.

Endocrine Disturbances of the Ovary.

SEE: p. 804.

Tumors of the Ovaries: Tumors may be benign or malignant.

Benign Tumors: These are cysts, solid tumors such as papilloma, fibroadenoma, fibroma, fibromyoma, angioma, lymphangioma, chondroma, and osteoma In this classification may also be included Brenner tumors and adrenal

tumors of the ovary and luteoma (masculinoovoblastoma).

Cysts of the Ovaries. These may be small or exceedingly large and may spring from various structures of the ovary, causing either endocrine disturbances or pressure symptoms because of the space they occupy in the abdomen.

Diagnosis: If the cyst is very small it may escape detection; if large it is easily palpated by bimanual examination; when very large it causes distention of the abdomen and crowding of the abdominal viscera Fluctuation may or may not be elicited.

Dermoid Cysts These may be unilateral or bilateral and, when large, may be palpated externally and bimanually X-ray examination may reveal the presence of teeth, hair, bone or other embryonic tissue.

Solid Tumors: These may be large or small, single or multiple They do not cause metastasis but, if large, may cause considerable discomfort and interfere with ovarian and uterine function They may be diagnosed by bimanual palpation The structure of the tumor can only be diagnosed by microscopy.

Brenner Tumors These are believed to be benign and are said to arise in the ovary from cell nests of Walthard They are of two kinds, solid and cystadenomatous. When they occur during the menstrual life no characteristic effect upon menstruation is noted; in older women it has been suggested that they may cause postmenopausal bleeding It is generally agreed that these tumors have no hormonal activity. When the tumor is large it may be diagnosed as a neoplasm, its morphology may be determined by microscopic examination.

Adrenoovarian Tumors (masculinoovoblastoma): This type of tumor is made up of adrenal tissue and develops within the ovary. It may spring from adrenocortical rests. It is unlike the Gravitz tumor or hypernephroma. At times pituitary, ovarian and adrenal tumors may coexist as individual entities These are often responsible for virilism or for Cushing's syndrome (See. pp. 765 and 805).

Malignant Tumors: These are carcinoma, adenocarcinoma and various other types of carcinomatous tumors, which may affect the various structures of the ovary such as the granulosa, the theca, the luteal cells, etc They may also be arrhenoblastoma and dysgerminoma These tumors are classified as embryonic or dysontogenetic. Other malignant tumors are chorionepithelioma, hypernephroma, teratoma, sarcoma of various types, melanoma and the Krukenberg tumor.

Carcinoma According to Curtis[1] approximately 20 per cent of ovarian tumors are malignant The commonest form is cystic carcinoma, generally known as papillary serous cystadenocarcinoma. The solid type of ovarian carcinoma is less common than the cystic form; the tumors may be medullary, scirrhous or adenomatous, and are often bilateral Carcinoma of the ovary may affect its endocrine structures or other parts; it may be primary or secondary The tumors may be of various sizes and may cause metastasis.

Hypernephroma: A hypernephroma usually develops from adrenal rests This type of tumor usually invades the kidney but may also affect other organs, particularly the ovary It may be primary in the ovary or it may metastasize to the ovary from hypernephroma of the kidney; it usually grows to a large size and,

[1] Curtis, A. H.: Textbook of Gynecology, p 305, Saunders, Philadelphia, 1938.

because of its structure, it has been classified by some authors as an adenocarcinoma.

Sarcoma: Sarcoma of the ovary is rare. It is often bilateral; frequently of the spindle cell variety. Endothelioma and perithelioma of the ovary are often classified as ovarian sarcoma.

Krukenberg Tumor: This is a special type of carcinoma of the ovary, generally bilateral, causing diffuse infiltration though preserving the normal contour of the ovaries. It is a secondary invader from the stomach or other parts of the gastrointestinal tract The microscopic picture of the tumor simulates that found in carcinoma of the stomach, *i. e.*, large, swollen, signet-ringlike cells buried in a connective tissue matrix and areas of mucoid degeneration.

Dysgerminoma These tumors originate from the undifferentiated embryonic gonadal cells and are responsible for the development of pseudohermaphrodites

Granulosa Cell Tumors These originate from the granulosa cells of the graafian follicle. They are responsible for precocious puberty as evidenced by early development of pubic hairs and premature menstruation.

Theca Cell Tumors These originate from the theca cells of the graafian follicle and usually occur in women beyond the menopause, causing the return of periodic bleeding, enlargement of the uterus and hyperplasia of the endometrium with an increase in the production of estrogen.

Arrhenoblastoma: These originate from male directed cell rests in the ovary. These tumors cause masculinization or virilism in previously normal women (SEE: p. 805).

Malignant tumors of the ovary may cause ***various endocrine changes*** depending upon which of the ovarian structures are invaded. These changes, as mentioned above, may be precocious matronism, virilism, masculinization or pseudohermaphroditism. They may also cause menstrual disturbances and sterility.

Examination of the Male Genital Organs

The male generative organs are examined by *inspection* and *palpation.*

The Penis: This is examined as to the condition of the prepuce, the presence of rashes, such as chancre, chancroid, condylomata, nonvenereal rashes, carcinoma, tuberculosis and also for the presence of scars, as they may denote healed lesions

The Urinary Meatus: This is examined for discharges and the position of the meatus should be noted, *i. e.*, whether it is in the normal position, on the undersurface (hypospadias), or on the dorsum of the penis (epispadias).

The Scrotum: This is examined as to size and the condition of the blood vessels Enlargement of the scrotum may be due to hernia, hydrocele, varicocele and orchitis A very small or rudimentary scrotum is found in eunuchoidism, pseudohermaphroidism and in cryptorchism (SEE: p 801).

The Testicles: These are examined as to size, number, consistency and position; they should be palpated for the presence of hard masses and for tenderness. Tuberculosis, carcinoma, mumps, various types of orchitis and syphilis may affect these glands.

The Spermatic Cords: The condition of the spermatic cords should be investigated as to size and tenderness

Malformation of the genitalia as well as the ***secondary sex characteristics*** of the individual should be noted.

The Inguinal Regions: These should be inspected and palpated for hernia and enlarged glands; the femoral ring should be palpated in order to determine its size. During palpation of the ring the patient is asked to cough, the strength of the impulse should be noted and also

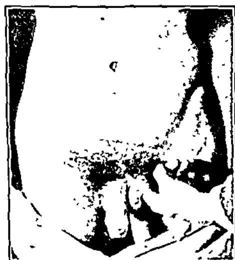


Fig 9—Technic for palpating for inguinal hernia

if there is any protrusion of viscera. In the presence of a hernia, one should determine whether it is direct or indirect, also if it is partially reducible, totally reducible or irreducible Finally, a most important procedure which is often overlooked, is the examination of the prostate gland

The Prostate Gland: No physical examination of middle-aged men is complete unless the prostate is investigated. The prostate is a firm, partly glandular and partly muscular body. It is situated in the pelvic cavity below the lower part of the symphysis pubis, in front of the rectum, and immediately below the internal urethral orifice and around the commencement of the urethra. The prostate is examined by the palpating finger through the rectum. Its size and consistency may thus be noted. The commonest disease of the prostate is hypertrophy and prostatism. It may also be the seat of neoplasms, calculi, tuberculosis, syphilis, inflammatory conditions, etc.

Diseases of the Male Genital Organs

Diseases of the male gonads may cause local manifestations or endocrine disturbances. (For Endocrine Diseases of the Gonads—SEE: p. 801.)

Diseases of the Penis

Congenital Deformities: ***Congenital Absence of the Penis:*** In this anomaly, the urethra may open anywhere on the perineum or on the anterior rectal wall The male secondary characteristics are not disturbed



Fig 10—Palpating for hernia during cough.

Double Penis: Two distinct and well-formed organs may appear in the place of one. In some instances this is associated with double bladder so that there is a penis for each bladder; in other in-

because of its structure, it has been classified by some authors as an adenocarcinoma.

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Malformation of the genitalia as well as the ***secondary sex characteristics*** of the individual should be noted.

The ulcers are usually multiple; they are at first round or oval, and later become irregular, ragged and superficial, having a gray base covered with a copious purulent discharge; they are soft and not indurated These lesions may cause considerable destruction of tissue, lymphadenitis and enlargement or suppuration of the inguinal lymph nodes. (SEE Fig 11)



Fig. 12—Granuloma of penis

Lymphogranulomatosis Inguinale (Lymphopathia Venerum). This is a chronic disease of venereal origin said to be due to a filtrable virus It occurs more frequently among women The initial lesion usually consists of several herpes on the glans around the corona or other parts of the penis. Several weeks later there develops inguinal adenitis; these glands suppurate and form abscesses and fistulae, often causing tissue destruction and rectal strictures. The Frei test is usually positive (SEE: pp 697 and 1055.)

Granuloma Inguinale (Granuloma Venereum, Serpiginous Ulceration of the Groin): This is a disease found chiefly in the negro, occurring more often among women and in the tropics (SEE: p 696). It is said to be due to the "Donovan Bodies" It is characterized by the formation of serpiginous, granulomatous ulcers of the skin and subcutaneous tissue of the penis and inguinal regions The ulceration frequently appears first in the groin and then spreads to the penis, scrotum and perineum. It is a chronic disease causing little discomfort aside from some itching and a foul discharge. (SEE: Fig. 12.)

Balanoposthitis (Erosive and Gangrenous Balanitis): This is sometimes spoken of as the "fourth venereal disease." It is a specific infection which,



Fig 13—Epithelioma of prepuce and glans penis (Courtesy, Dr. Costello, Philadelphia General Hospital)

according to Herman[1] is due to a spirochete growing in symbiosis with a vibrio. The normal habitat of the organism is in the mouth, being transferred to the penis by the saliva The disease is comparatively rare The lesions first manifest

[1] Herman, Leon "The Practice of Urology," p 581, W B Saunders Co, Philadelphia, 1938

stances there is but one bladder and urine may be passed by both organs.

Epispadias: This is a rare condition in which the urethral opening is situated somewhere along the dorsum of the penis; it may be associated with partial incontinence. *Epispadias totalis* is accompanied by extrophy of the bladder, wide separation of the pubic bones, cryptorchism and other deformities.

Hypospadias: This is a common anomaly; the urethral opening is usually situated medially anywhere along the undersurface of the penis.

Phimosis: This is a congenital contraction of the preputial orifice It is generally associated with elongation and hypertrophy of the prepuce and an inability to retract it over the glans during erection. Phimosis may be congenital, or acquired because of injury, inflammatory disease or edema.

Paraphimosis · This denotes strangulation of the penis after the prepuce had been retracted over it so that the foreskin cannot be brought forward.

Venereal Diseases of the Penis: ***Chancre (Hard Chancre):*** This is the initial lesion of syphilis appearing three to six weeks after exposure. It is manifested first as a papule and later as a punched-out ulcer having a hard base It is indurated, clean and painless, and is usually single, though multiple chancres are not rare. It may appear anywhere upon the penis; the site of the lesion often modifies its appearance. In the coronal margin it appears as a superficial erosion; in the coronal sulcus it usually develops into a large ulceration; at the preputial margin and on the frenum, it appears as indurated fissures; on the glans it is a superficial indurated craterlike punched-out lesion with clean-cut edges having a red base covered with a grayish exudate. The cause of chancre is treponema pallidum which may be recovered from the scrapings of the ulcer. The sero-diagnostic tests for syphilis do not become positive until several weeks after the chancre has appeared. Bilateral enlargement of the inguinal glands develops a short time after the appearance of the chancre.



Fig 11—Chancroid and abscess of penis.

Mucous Patches (condyloma lata): These are slightly raised, moist grayish white patches; they are one of the manifestations of secondary syphilis and may appear upon the penis or elsewhere in association with other secondary lesions

Gumma: Gumma of the penis is rare; it is a tertiary manifestation of syphilis. It may appear as a large circular ulcer with steep sides having a punched-out appearance.

Chancroid (soft chancre): This is caused by the Ducrey-Unna bacillus The lesion is an ulcer appearing on the genitals within several days after exposure

prolonged irritation of the penis or prostate. Priapism occurs fairly frequently in association with leukemia.

Diseases of the Urethra

Venereal Diseases of the Urethra: ***Gonorrhea:*** Gonorrheal urethritis is the commonest infection of the urethra. It is caused by infection with the gonococci. Acute gonorrhea is characterized by inflammation of the external urinary meatus and chiefly by the discharge of pus which contains the gonococci. In mild cases infection is limited to the anterior urethra. In severe cases infection may spread to the posterior urethra.

Chronic Urethritis: This is usually the result of acute gonorrheal urethritis and is manifested as an organic stricture. The symptoms are chronic mucopurulent discharge, shreds in the first or second specimens of urine, or in both, and occasional prostatic complications. Occasionally it may be of nonspecific origin.

Syphilitic Urethritis: This is caused by an intraurethral chancre. It is characterized by its prolonged incubation period, scanty seropurulent discharge and bilateral inguinal adenopathy. Occasionally both syphilitic and gonorrheal urethritis may occur at the same time, since both are procurable in the same shop.

Nonspecific (nonvenereal) Urethritis: This may be caused by a variety of organisms, *i. e.*, the staphylococci, the trichomonas vaginalis or other organism which may enter the urethra during sexual intercourse or from filthy habits. Nonspecific urethritis may also result from injury to the urethra by trauma, catheterization, or from foreign bodies in the urethra. The symptoms are tenderness, burning on urination, and occasionally a serous discharge.

Diphtheritic Urethritis: This is characterized by intense inflammation of the urethra, a serosanguinous discharge and the formation of a membrane which may be visible in the meatus. A culture taken from the urethra may disclose the diphtheria bacilli.

Spermatorrhea: This is characterized by the discharge of a clear, glycerine-like discharge, usually during erection. It may be due to overfilling of the seminal vesicles or the prostate. It is not a urethritis, but may be mistaken as such unless the secretion is examined microscopically.

Other Types of Urethritis: These may be due to foreign bodies, neoplasms, various other infections and parasites in the urethra.

The symptoms of nearly all types of urethritis are burning on urination, often frequency of urination, some pain and tenderness over the penis and urethra and a urethral discharge varying in consistency and content depending upon the cause of the urethritis.

Diseases of the Scrotum

Congenital Malformation of the Scrotum: The scrotum may fail to develop as seen in bilateral cryptorchism and in some of the anomalies of the penis, testes and urethra.

Bifid Scrotum: This is a distinct division of the scrotum into two lateral halves. It may be mistaken for a vulva, particularly when associated with hypospadias or with a poorly developed penis.

Acquired Lesions of the Scrotum: The scrotum may be affected by various skin lesions, parasites, tumors, edema, hydrocele, varicocele and hernia.

Skin Lesions of the Scrotum: There may be dermatitis such as eczema, intertrigo, erythema, etc.; they affect the

themselves as white superficial patches surrounded by an inflammatory zone which suppurates and discharges a yellowish white seropurulent pus having a foul odor. The ulcers may be superficial and circumscribed or they may cause gangrene and destruction of the prepuce, glans and shaft of the penis



Fig 14—Carcinoma of penis

Malignant Lesions of the Penis: ***Carcinoma:*** The lesion is usually an epithelioma of the papillary, vegetative or cauliflower, or the ulcerative type Rarely there may be a melanotic or a medullary type of carcinoma. The lesions may start as a papilloma or as an ulcer with slightly raised edges, which causes ulceration and inflammation and, eventually, destruction of the penis The lesions may be primary or secondary, usually causing metastasis. (SEE: Figs. 13 and 14.)

Sarcoma: Sarcoma of the penis is rare; it may occur on any part of the penis and cause obliteration of the cavernous space which may give rise to priapism.

Tuberculosis of the Penis: In the adult it may be due to direct infection during sexual intercourse from a tuberculous vulva, or it may be secondary to tuberculosis along the genitourinary tract. The lesion may start as a single focus which ulcerates slowly; it is irregular in contour and depth and may be covered with granulation tissue and slough. The lesion may heal spontaneously or it may cause severe ulceration of the penis (SEE: Fig. 15).

Benign Lesions of the Penis: These may be venereal warts, dermoid cysts, angiomata, fibromata, lipomata and various skin lesions such as herpes simplex or herpes zoster, lichen planus, scabies, abrasions, etc.

Priapism: This is a condition of continuous penile erection not due to sexual emotion The erection may last from several days to several months or longer. It is often attended with pain but without

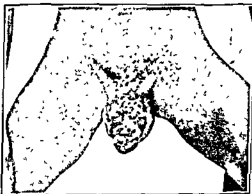


Fig. 15—Tuberculosis of the penis. (Philadelphia General Hospital)

libido; sexual intercourse aggravates the condition. Priapism may be caused by: Thrombosis of the cavernous bodies (or thrombosis may cause it); sexual excess; injury; neoplasm; myelitis; fracture of the spine; *tumor of the cord;* syphilis of the cord; urethral stricture;

folds of the skin, causing itching and burning, and occur chiefly during the summer. Erythema may be caused by chafing as the result of irritation or infection by various fungi, *i. e.*, ringworm, dermatophytosis, or by the streptococcus pyogenes.

Tinea Cruris (dhobie itch, jockey-strap itch, red flap): This occurs upon the upper and inner parts of the thigh and extends to the scrotum, perineum and anus. It is caused by the Epidermophyton inguinale, a fungus closely related to the trichophyton. It is characterized by the formation of an erythematous and scaling or vesicular and crusted patch which spreads peripherally and clears in the center, having a well-defined border, particularly at its lower edge. Other yeast fungi may affect the same region.

Pediculosis Corporis (crabs): This may affect the mons, scrotum, thigh or any hairy surface They cause intense itching

Pruritis: Itching of the scrotum may occur in the various skin affections and parasitic infections, or it may be caused by boils, anal and urethral discharge, or by worms It is also found in diabetes, tuberculosis, renal disease, and in the bedridden who have incontinence of urine, and have profuse sweating; occasionally it may occur idiopathically.

Tumors of the Scrotum: These may be vascular such as nevi or hemangioma; or solid tumors such as fibroma, lipoma, sarcoma, chondroma, osteoma and teratoma.

Inflammation of the Scrotum: This may result from wounds, and other trauma, or it may extend from orchitis.

Gangrene of the Scrotum: This may result from infection from syphilis and from irritation caused by the dribbling of urine over the scrotum for extended period.

Edema of the Scrotum: This m be found in general anasarca caused heart failure, in nephritis or *in oth* conditions that cause edema. The scr tum and penis may become enormous enlarged and have a doughy feel; t skin is thick and pale, *it is not tender*, and does not itch.

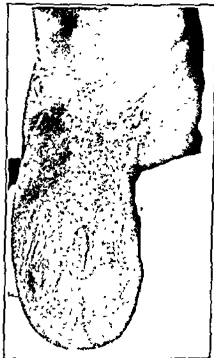


Fig. 16—Hydrocele
(Philadelphia General Hospital.)

Angioneurotic edema is rare, but does occur occasionally. Part of the scrotum becomes swollen, red and hot; the swelling is accompanied by severe itching or burning.

Elephantiasis of the Scrotum: Elephantiasis of the scrotum may be due to lymphatic obstruction, to lymphopathia venereum, and to the invasion of the lymph nodes by the filaria sanguinis homonis.

Hydrocele: This may be congenital or acquired; it is characterized by the accumulation of fluid in the tunica vaginalis testes, or in the processus vaginalis. The scrotum may become enormously distended and may be mistaken for hernia. It yields a dull note on percussion; it may fluctuate, is not tender, is irreducible, and transmits light. The skin of the scrotum is stretched but is otherwise normal. (SEE: Fig. 16.)

Lymphocele: This is an accumulation of lymph in the scrotum It may be due to rupture of dilated lymph vessels or to filariasis.

Hematocele: This is an accumulation of blood in the scrotum. It may be caused by trauma such as a blow or a wound, or by puncture of a blood vessel following the tapping of a hydrocele. It may also be due to spontaneous rupture of a blood vessel, or to hemophilia or purpura.

Disease of the Testes

Endocrine Disturbances of the Testes (SEE: p. 801).

Congenital Defects of the Testes: The testes may be entirely absent (*anorchism*); one or both may be intra-abdominal, in the inguinal canal, on the perineum, or underneath the mons (*cryptorchism*); or they may be supernumerary (*polyorchism*).

Atrophy of the Testicles: This may be congenital or acquired because of trauma or disease.

Acquired Diseases of the Testes: *Orchitis* (Inflammation of the Testicle): This may be caused by trauma, by gonococci or other infections, by mumps, by tuberculosis, and by syphilis, and it may occur as a complication in infectious diseases.

Orchitis caused by trauma, infection or by mumps is characterized by extremely painful swelling and enlargement of the affected testicle. It is associated with fever, and the inflammation usually extends to the epididymis.

Epididymitis: Inflammation of the epididymis may be gonorrheal or non-specific, acute or chronic.

Gonorrheal Epididymitis: This may follow acute anterior gonorrheal urethritis when the infection extends to the posterior urethra, or it may be caused by chronic posterior urethral or prostatic infection. One or both epididymi may become affected; the inflammation may extend to the scrotum, the orchis and the spermatic cord. The *symptoms* are severe pain in the testes, swelling and tenderness in the affected epididymis, usually at the globus minor, and pain in the groin. The scrotum is thickened, inflamed and tender and there may be a hydrocele. The patient, when walking, is bent forward with legs spread wide apart and attempts to support the heavy and inflamed scrotum. The local inflammation is accompanied by a systematic reaction of fever and leukocytosis, and there may be an active gonorrheal discharge from the urethra

Nonspecific Epididymitis: This may be acute or chronic. It may be a result of direct trauma or it may occur as a complication in typhoid fever, meningitis, pyemia, and other febrile diseases. It may also be caused by posterior urethral inflammation incident to catheterization, or the introduction of a sound or other instrument Surgical operation upon the prostate or the lower genitals may cause epididymitis. Milder cases may be caused by prolonged sexual excitement without gratification. The *symptoms* are pain in the scrotum, perineum and groin Generally the inflam-

changes in the male are not as well-defined nor as perceptible as those occurring in the female, because there are no external visible signs, such as the menstrual phenomenon. The climacteric in the male is a slow process; it is more insidious in its onset and more gradual in its termination. The beginning of the climacteric period in men is somewhere between the fiftieth and fifty-fifth year There is usually a change in temperament, and the person becomes more emotional, fatigues easily, and sleep is often disturbed The appetite becomes irregular, and digestive and bowel irregularities develop The memory becomes less acute The sexual function gradually diminishes, and occasionally sexual perversion develops, because the libido is greater than the potentiality. In many instances the prostate begins to hypertrophy. Cardiac palpitation, or other functional irregularities, and heart consciousness with occasional chest pains, develop during emotional disturbances Hair over the body and pubes becomes scarcer, though hair in the external auditory canal, in the nasal passages, and at the eyebrows becomes more profuse The skin over the body develops atrophic changes, and fat disappears from in front of the neck so that the skin hangs in folds. The climacteric in men is so definitely associated with general aging that a time limit for its termination cannot be accurately determined At times it is difficult to judge the dividing line between the end of the climacteric and the beginning of senility However, among the majority of healthy men, there is a fairly large span between the time of cessation of sex function and the onset of senility During this interval, many of the nervous manifestations that were previously present now disappear, and the person becomes more tranquil and is capable of much mental and physical work and of enjoying an orderly and pleasant existence

ing in the axis of the organ. Its chief debility is the interference with coitus

Reiter's Disease (Infectious Urethritis, Ruhrrheumatismus, Spirochetosis Arthritica): Reiter's disease is a syndrome characterized by a triad of urethritis, arthritis, and conjunctivitis of unknown etiology. It occurs most frequently among young male adults and bears no relation to sex contact.

Reiter's disease is ushered in with an acute mucopurulent urethral discharge As the disease progresses, the anterior urethral inflammation may extend to the posterior urethra, the prostate, and the bladder, and superficial ulcerations may appear on the glans penis. Smears and cultures of the urethral discharge are negative for gonococcus Within two weeks after the onset of the urethritis, polyarthritis and conjunctivitis make their appearance. These may appear simultaneously, or one may precede the other The joints affected become red, hot, and swollen, and are exceedingly painful Any of the joints may become affected Occasionally effusions may develop within an affected joint, and osteoporosis may occur at the ends of the bones. The conjunctivitis is diffuse and has a purulent discharge. The inflammation may spread to the scleras and corneas.

There is usually a moderate rise in temperature, between 100 5° to 101 50° F, a moderate leukocytosis (10,000 to 12,000), and a rapid sedimentation rate The disease may last from two to six months and has a tendency to recur.

Complications such as keratosis, general lymphadenopathy, gastrointestinal disturbances, and cardiovascular changes may occur from time to time. Permanent arthritic changes are rare

Puberty and Climacteric in the Male: Puberty in the male occurs somewhat later than it does in the female. In the temperate climate, the onset of puberty is usually between the ages of thirteen and fifteen years During this period, hairs begin to sprout upon the upper lip and chin, and some hair appears upon the pubes The testes, scrotum, and penis enlarge and develop, and there is a spurt of bodily growth The prostate usually develops as a functioning organ by the sixteenth year With the development of the prostate, the genitalia become sexually potent, the voice thickens, and the distribution of the bodily hair assumes male characteristics. A secondary spurt in bodily growth generally occurs between the ages of eighteen and twenty-one years, just before epiphyseal ossification

Precocious Puberty (Macrogenitosomia Praecox): This is a condition in which sex maturation occurs before the normal age of its usual appearance It is characterized by premature development of the sex organs and of the secondary sex characteristics This may occur at any age preceding the age of normal puberty. Precocious puberty may be caused by a pineal tumor, by a tumor or hyperplasia of the basophilic cells of the pituitary gland, or by hyperplasia or tumor of the adrenal cortex

The Male Climacteric: The climacteric in either the female or the male is a period of readjustment to a developing imbalance between the gonadal and the pituitary sex hormones. In the male, the testicular hormones gradually diminish, while the formation of the pituitary sex hormones either continues as during the preclimacteric era, or is actually increased In either case, there exists a disproportion between the testicular and pituitary hormones. During this period of readjustment, certain psychic and somatic changes become evident. These

SECTION 11

Bones and Joints

CHAPTER XXV

Examination and Diseases of the Bones and Joints and of the Extremities

The examination of the extremities, including their bones and joints, is a part of every general physical examination.

Much may be learned by a careful examination of these members of the body as it may reveal developmental errors, birth injuries, childhood bone and nerve disease, and such adult injuries and diseases as have a predilection for the bony structures, joints or the soft parts of the extremities

At present, most examinations of the bony framework of the body are considered incomplete unless checked by the roentgen ray. To interpret a roentgenogram correctly, one must have a thorough knowledge of the normal structure, and the various changes that may occur in a given area as a result of disease. Therefore, a thorough physical examination is essential for a correct diagnosis, which can be amplified and confirmed by the x-ray findings.

Ossification Centers

In the normal, ossification centers and epiphyseal union of various bones should occur at definite ages. Marked deviation from the normal indicates a pathologic process. Bone development at various ages and the appearance of ossification centers and epiphyseal union show the following·

At Birth: Both fontanels are open; ossification centers are noted in the lower end of the femur, the head of the tibia and some of the bones of the foot (astragalus, calcaneus, and cuboids)

At Two Months: The posterior fontanel closes; the first ossification center is noted in the head of the humerus.

At Six Months: The two lower central incisors of the deciduous set appear between the sixth and eighth month. Centers of ossification are noted in the lower end of the radius, the lower end of the tibia, the os magnum, and the unciform bones of the wrists.

At One Year: The four upper incisors are erupted; ossification centers are now found at the head of the femur and the third tarsal cuneiform bone.

At Two Years: The four canine teeth are erupted and the anterior fontanels are closed. The usual closing time is at or about 1½ years Ossification has already occurred in the upper scapula, the lower end of the humerus, the pyramidal bone of the wrist, and the second center for the head of the humerus.

At Three Years: The four posterior molars are erupted; ossification is noted at the extremities of the metacarpal, metatarsal, and phalangeal bones.

At Four Years: There is ossification of the semilunar bone (wrist) the head of the fibula, the scaphoid, and the first and second cuneiform bones of the foot.

At Five Years: There is ossification of the head of the radius, the scaphoid, the semilunar bones of the wrist, the patella, and the greater tuberosity of the femur.

At Six Years: The first molars of the permanent teeth usually erupt and epiphyseal junction occurs at the head of the humerus.

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Ossification Centers

In the normal, ossification centers and epiphyseal union of various bones should occur at definite ages. Marked deviation from the normal indicates a pathologic process. Bone development at various ages and the appearance of ossification centers and epiphyseal union show the following:

At Birth: Both fontanels are open; ossification centers are noted in the lower end of the femur, the head of the tibia and some of the bones of the foot (astragalus, calcaneus, and cuboids).

At Two Months: The posterior fontanel closes; the first ossification center is noted in the head of the humerus.

At Six Months: The two lower central incisors of the deciduous set appear between the sixth and eighth month. Centers of ossification are noted in the lower end of the radius, the lower end of the tibia, the os magnum, and the unciform bones of the wrists.

At One Year: The four upper incisors are erupted; ossification centers are now found at the head of the femur and the third tarsal cuneiform bone.

At Two Years: The four canine teeth are erupted and the anterior fontanels are closed The usual closing time is at or about $1\frac{1}{2}$ years. Ossification has already occurred in the upper scapula, the lower end of the humerus, the pyramidal bone of the wrist, and the second center for the head of the humerus

At Three Years: The four posterior molars are erupted; ossification is noted at the extremities of the metacarpal, metatarsal, and phalangeal bones

At Four Years: There is ossification of the semilunar bone (wrist) the head of the fibula, the scaphoid, and the first and second cuneiform bones of the foot.

At Five Years: There is ossification of the head of the radius, the scaphoid, the semilunar bones of the wrist, the patella, and the greater tuberosity of the femur

At Six Years: The first molars of the permanent teeth usually erupt and epiphyseal junction occurs at the head of the humerus.

At Seven Years: The incisors begin to erupt at the seventh year and are fully erupted by the eighth year; ossification at the lower end of the fibula is completed by the end of the seventh year.

At Nine Years: Ossification is noted in the olecranon process, the lesser tuberosity of the femur, and the head of the os calcis.

At Ten Years: The eight bicuspids should be erupted, and the external condyle and the pisiform bones should be ossified.

At 11 Years: The canine teeth begin to erupt at 11 years and should be fully erupted by the fourteenth year. Ossification is noted of the internal condyle, the trochlea and the head of the tibia (second center).

At 12 Years: The second molars begin to erupt at 12 years and should be completely erupted at 15 years

At 13 Years: Signs of puberty should be well marked. Ossification is completed at the head of the acromion process, the tip of the scapula, and the outer end of the clavicle. Epiphyseal junction is noted at the head of the calcaneum

Epiphyseal Junctions: Epiphyseal junctions of the various bones occur at different ages. The head of the humerus, first ossification center, at two months, second at six years; the head of the calcaneum at 13 years; the olecranon at 14 years; the trochlea and the head of the radius at 15 years, the tuberosity of the femur at 16 years; the internal condyle at 17 years; the acromion process, the outer end of the clavicle, the heads of the metacarpal, metatarsal and phalangeal bones, the head of the femur and the lower ends of the tibia and fibula at 18 years, the lower end of the femur and the heads of the tibia and fibula at 19 years; the second center for the head of the humerus, the tip of the scapula, the external condyle, and the lower end of the radius at 20 years; and the lower end of the fibula at 21 years. By the end of the twenty-first year, ossification and epiphyseal union should be completed.

Premature ossification occurs in hypergonadism. Delayed ossification is seen in hypopituitarism, hypogonadism, hypothyroidism and in gigantism. The bones are thinner than normal in hyperthymism

The Bones

Physical Examination of the Bones

The parts to be examined as well as the corresponding parts of the body not under examination must be bare of clothing so that the two parts may be carefully compared. This is done by *inspection, palpitation, manipulation, mensuration* and *auscultation* and often by *x-ray examination.*

By *inspection* the patient's posture may be studied, and this should be done while he is lying, standing, walking and stooping, every aspect being minutely observed The posture of the body as a whole and the extremities may be thus studied, and any atrophy of the muscles, tumefactions or distortions of the joints, angles of bones, or curvature of the spine should be noted and estimated. The *color* of the parts and the existence of dilated veins should also be observed

By *palpation* the muscles are investigated as to their rigidity or flabbiness; the joints are felt in order to note if they are rigid, relaxed, hard, soft or brawny; enlarged glands are thus dis-

covered; bony prominences outlined, and displacements ascertained.

By *manipulation* the condition of the joints may be determined, *i. e.*, whether the joints are limited in range of motion, rigid or in a healthy condition.

Mensuration is a most valuable means of determining the definite degree of any existing deformity, and by keeping records and comparing them from time to time, it can be determined whether the condition is improving, is stationary or is growing progressively worse

By *auscultation*, now little practiced, *the early orthopedists recognized five* sounds: (1) Simple, dry friction sound; (2) dry grating sound, (3) coarse grating sound; (4) moist crepitant sound, (5) coarse crepitant sound (McCurdy).

X-ray or roentgenographic examination will reveal deformities, fractures, bone densities and calcific deposits

Bone Diseases[1]

The bones in general are studied with a view to determining their size and shape. The bones of the body may be deformed because of disease, or such deformity may be caused by (I) Injury; (II) infectious diseases, (III) general disease, not limited to one bone, (IV) tumors, and (V) cysts

I. **Injury:** An injury may cause localized swelling by producing subperiosteal hemorrhage, by the formation of callus at the site of a fracture, or by a deformity due to a poorly-united fracture

These may be recognized by inspection and palpation and by an x-ray examination. A subperiosteal hemorrhage usually presents an elevation which is tender to touch, somewhat yielding to deep pressure and, when not under great tension, may give rise to fluctuation. This may be elicited by gently tapping simultaneously with the flexor sides of the distal phalanges of both index fingers at the divergent limits of the swelling The presence of callus at the site of a fracture is recognized by the presence *of an abnormal elevation along an otherwise* smooth surface of a bone. The elevation is hard and nonyielding to touch and is usually painless Bone deformity due to a badly united fracture may be diagnosed by a change in the general contour of the bone at a certain point, which may result in angulation or other

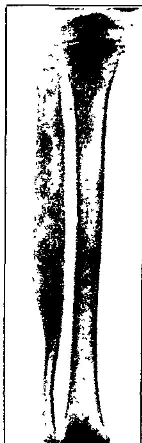


Fig 1—Osteomyelitis
(Courtesy of Dr. Leon Solis-Cohen.)

[1] For Symptoms, See p 81

deformity, often interfering with normal function.

II. Infectious Diseases: Infectious *diseases may give rise to inflammatory* changes in the bone; if the initial inflammation is in the periosteum, *periostitis* will take place; but if the bone structure is affected, *osteomyelitis* may result Acute infection may occur in a bone because of direct injury, or indirectly by

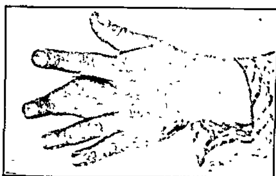


Fig 2—Tuberculous dactylitis. (*Spina ventosa.*)

the infection being carried to it by the circulation from a remote portion of the body.

The presence of periostitis and osteomyelitis is recognized by the occurrence of pain over the affected part and by fever, sweats and leukocytosis Pressure over the affected part causes pain; the overlying muscles are usually rigid and the skin may become inflamed. When suppuration occurs, fluctuation may be elicited.

Bone Tuberculosis (Tuberculous Osteomyelitis): This usually starts in the cancellous ends of long bones and has a tendency to spread to the epiphysis, often invading the joint; occasionally the shaft may become involved This disease is not confined to the long bones. The common sites of infection are the vertebrae, the lower end of the femur, the pelvis, the hips, the tibia and fibula, the foot, the bones of the head and face, the sternum, the humerus, the radius and ulna, the fingers and the scapula. The patella is rarely affected.

Symptoms: During the early stages there may be fever, malaise, and some pain over the affected part. When necrosis and suppuration develop, there may be fluctuation, signs of bone destruction and the formation of discharging sinuses; often the pus may burrow its way along the sheaths of muscles or large vessels and form a cold abscess at a distance from its seat of origin

Tuberculous dactylitis occurs principally in the young. When the shaft of the bone is affected, causing periosteal swelling, it is called *spina ventosa*

Osteomyelitis: This is an inflammation of the bone marrow cavity affecting the soft tissues and the cells in the Haversian canals in the cancellous spaces or in the medullary cavity. It may be simple or infective; either type may be acute or chronic, localized or diffused.

Simple osteomyelitis is not due to bacterial infection. The localized form is caused by traumatism, *i. e*, contusion or fracture. The diffuse variety is often seen in conjunction with rickets or osteitis deformans. It usually causes softening of the bone and permits bending. The chronic type causes sclerosis of the bone

Infective or pyogenic osteomyelitis is caused by bacterial infection, *i e*, the staphylococci, streptococci, pneumococci, typhoid and paratyphoid bacilli, tubercle bacilli, gonococci and various pyogenic organisms The infection may be carried by the blood stream, by the lymphatics or it may gain entrance through a wound

The clinical manifestations are high fever, chills and sweats; this may be ac-

companied by prostration. Pain is acute, boring, gnawing or aching over the affected area and there may be marked tenderness on palpation and on manipulation. Swelling, distended veins and edema develop later. During the early stages the x-ray may not reveal the affected area. When necrosis develops, the x-ray examination may indicate it.

Periostitis: This may be acute and chronic. The inflammation is seldom confined to the periosteum alone, but generally occurs in conjunction with inflammation of the bone (osteitis), thus causing an osteoperiostitis.

Acute periostitis is often associated with some degree of osteomyelitis It may be caused by trauma, extension of inflammation from other structures, blood stream infection, certain febrile diseases, exposure to cold, constitutional diseases, by poisoning with phosphorus or mercury. It is often found among pearl polishers. This condition may affect any bone, it may be localized or diffused.

Symptoms There is a sharp rise in temperature, severe pain worse at night, and exquisite tenderness to touch over the affected area, which is red and hot to the touch

The disease may terminate in resolution or it may cause bone necrosis

Chronic periostitis is often syphilitic and may be manifested by the formation of nodular swellings. These are usually soft and not very tender to touch. Occasionally they may degenerate and involve the bone, causing caries or necrosis.

Syphilis: This may be acquired or congenital. *Acquired syphilis* is characterized by periosteal thickening, and, in the tertiary stage, by gummata. In *congenital syphilis*, bone swellings are quite common and periosteal thickening of the skull bones is frequently noted (Parrot's node)



Fig 3—Sabre-shaped tibia.

According to Goldthwait, the bone lesions of syphilis occur most commonly in the hereditary form and in the tertiary stage of the acquired disease, being rare in the secondary stage, though acutely sensitive small areas of periostitis, often multiple, are occasionally found in the secondary stage.

Hereditary lesions are divided into *early* and *late* forms. The *early form* occurs soon after birth and resembles rickets. Gelatinous masses are formed beneath the periosteum and at the epiphyseal line with sometimes true fracture of the shaft or separation of the epiphyses. There is said to be thickening of the periosteum and bone cortex. The so-called "juxta epiphyseal type" of late hereditary syphilis displays areas of

bone necrosis in the diaphysis with calcareous deposits beneath the periosteum and cortical thickening. The existence of this overgrowth, the fact that the joint surfaces are, as a rule, unaffected, and the location of the lesions in the diaphyses, differentiate this condition from diffuse tuberculosis. The absence of high temperature and the comparative mildness of the symptoms, in proportion to the extent of bone involvement, differentiate it from osteomyelitis

In the *late hereditary and tertiary forms*, cortical thickening and calcareous or show only slight hyperostosis. Motion is nearly normal, but there is considerable muscular atrophy.

(*b*) Those *in which bony changes are most marked*. Here is found marked hyperostosis; the end of the bone becomes enlarged, smooth, and rounded. The development may be rapid. Pain, if present, is *worse at night*. This type, the least amenable to treatment of all types, is usually associated with severe manifestations of the disease. It may result in ankylosis or great relaxation and hypermobility. *If secondary infec-*



Fig 4—Syphilitic knee joint. (Charcot's joint.)

deposits beneath the periosteum are the most characteristic and common manifestations of lues. These give rise to the typical *sabre-shaped tibiae* and the tender, fusiform swellings along the shafts of the long bones which are so well recognized.

Later lesions may be classed under two heads:

(*a*) Those in which the *synovial lesions predominate* are most common in the knee and are usually nonarticular There is marked effusion, a gradual onset, and often sharp pain On examination, rounded or flattened firm bodies are discovered located at the points of ligamentous attachment. They are somewhat movable and resemble foreign bodies. These are gummata, and may go on to ulceration and open into the joint. The bony ends are usually intact

tion occurs and sinuses form, the periosteum may react and produce new bone with cavities.

In general, serous and serofibrinous arthritis may occur in the early stages of syphilis, whereas gummatous infiltration is more characteristic of the later course of the disease.

Periosteal Nodes or Abscesses: These may be caused by syphilis, by an injury and often occur as a complication or sequel to typhoid fever and other infections

These may affect the bones of the skull or other bony structures. The nodes are strictly localized, seldom spreading beyond the involved bone. The diagnosis is based upon the finding of one or more hard nodes which later may undergo softening. It is usually unattended by sharp pain and local signs

of inflammation. The history is often of great diagnostic value.

III. General Disease of the Bone: ***Rickets:*** This is a nutritional disease occurring in infancy and early childhood due to a vitamin D deficiency which causes a disturbance of calcium and phosphorus metabolism which renders the bones more plastic. The bony growth is disturbed by changes at the epiphyseal lines; these changes are expressed by swellings that are externally visible and by irregular zones of proliferation The joints are unaffected except by the strain caused by faulty weight bearing. Before the bone deformities occur, the diagnosis of rickets is sometimes rather difficult Profuse sweating, a rather large head conveying the impression of squareness with its fontanels, especially the anterior, abnormally patent are always suspicious. Areas in which the skull seems thinned or softened are often palpable (Goldthwait).

The bone changes characteristic of rickets have been extensively discussed by J. S. Stone.[1] Enlargements at the epiphyseal lines, especially at the wrists and, to a less degree, at the ankles, will be noted on inspection and palpation The swelling at the junction of the ribs and their cartilages produces the rachitic "rosary," a row of beadlike nodules which can be palpated upon the ribs at their junction with the cartilages. The thoracic deformity commonest in rickets is the caving-in of the lateral chest walls and the protrusion of the sternum, commonly termed *pigeon breast*, or from its resemblance to the shape of a boat's hull, *pectus carinatum* When the tip of the ensiform cartilage is much depressed, what is termed *cobbler's chest* may sometimes be observed; very rarely the costal cartilages will dip in toward the sternum in such a way as to form a sulcus, producing a *gutter chest* (SEE p 239 for *Rachitic Chest Deformities*).

The abdomen is prominent and distended. The liver and spleen are often enlarged and the lower ribs flaring in consequence.

Diagnosis: In addition to the changes described in the bones and liver, rickets presents a fairly constant symptomatology, such as delayed dentition, delay in walking and talking, extreme irritability general body tenderness, restlessness, rolling of the head from side to side when in the recumbent posture, or a tendency to bury the occiput into the pillow There is profuse sweating about the head and neck and generally a slight but constant increase in temperature The child may be either thin with a large abdomen, or fat, flabby and of pasty appearance The urine contains an excess of calcium while the blood shows a deficiency of either calcium or phosphorus or of both X-ray examination may show that the epiphyseal line in the long bones becomes broad, concave and irregular The diaphysis appears ragged, it is broad and often cupped Most of the bones are less dense than normal.



Fig. 5—Rickets.
(Courtesy of Dr. Leon Solis-Cohen)

Osteitis Deformans or Paget's Disease: This is an affection occurring in adults. The etiology is obscure. It is slow and insidious in its onset. At the beginning of the disease the most com-

[1] Stone, J. S., Trans Amer Orthop Assn, 11, 337

mon complaint is pain in the lower limbs; the tibiae seem to be the bones most often affected. The deformities consist of thickening and bowing of the bones. The bowed appearance is usually due to the fact that the cortex thickens much more upon one side than upon the other. The medullary canal is sometimes completely hidden by trabeculae of bone, the bone being greatly diminished in density and weight. In some cases the cranium shows the earliest changes Goldthwait speaks of "acutely sensitive, swollen areas, exquisitely tender with the skin over them somewhat reddened," which never open spontaneously, but



Fig. 6—Rickets.

when incised do not exude pus, showing only chronic inflammatory tissue. The bowing of the legs often becomes so marked that the patient is forced to adopt a "scissors-leg" gait, the motion at the hip joint being greatly impeded.

While it is difficult to diagnose Paget's disease in its *early stages except by x-ray examination, it is readily* recognizable in the advanced stage The face seems small and triangular in shape

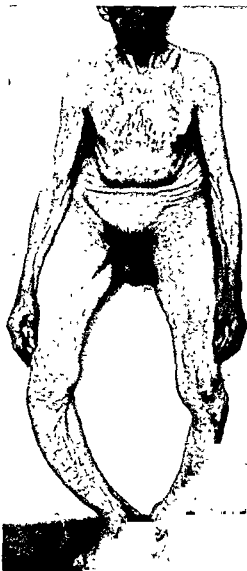


Fig 7—Osteitis deformans (Paget's disease). (*Jefferson Hospital.*)

with its base upward. The head is large and dome shaped, the upper dorsal vertebrae curve outward so that the head is pushed forward, the upper extremi-

ties seem to reach very low, resembling those of a chimpanzee. The legs and spine may become extremely bowed, so that the person becomes shorter in stature and develops an awkward gait

Paget's disease may at times affect only a few bones, either the head alone or one or both tibiae, fibulae or the femurs. In any case when there is reason to suspect its existence, recourse should be had to x-ray examination, which may show longitudinal striae of increased porosity and density in the same bones The skull bones are uniformly enlarged and show an irregular knobby appearance.



Leontiasis Ossium: A rare condition in which a general overgrowth of cranial and facial bones exists, causing a lionlike facial expression, accompanied by enlargement of soft parts of face and neck

Osteitis Fibrosa Cystica or Hyperparathyroidism: This is an inflammatory disease of the bones, causing a rarefying osteitis with fibrous degeneration and the formation of cysts The large, long bones are usually affected, *i e*, the femur, humerus and tibia. This condition is due to hyperparathyroidism which causes hypercalcemia and hypophosphatemia (SEE: p. 789).



Fig 9—Osteitis fibrosa cystica Note fractures of thigh and leg

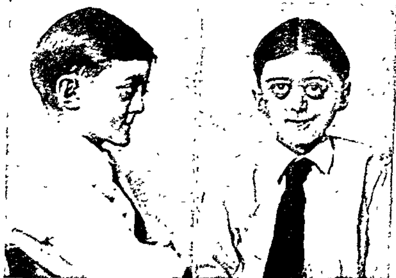


Fig. 10—Hand-Schuller-Christian's disease. Photograph of boy, age 10½ years (After Thompson, Keegan, and Dunn.)

Fragilitas Ossium: This is a disease of the bones associated with abnormal brittleness resulting in pathologic fractures. This condition may occur in either prenatal or postnatal life. It is usually associated with a peculiarly shaped head and blue sclerotics.

Senile Osteoporosis: This is the type of bone rarefaction seen in the aged. Pathologic fractures may result from



Fig 11—Hand-Schüller-Christian's disease (After Thompson, Keegan, and Dunn.)



Fig 12—Osteoma of phalanx

minor injuries or fractures may occur spontaneously without any injury. Rarefaction of the pelvic bones and of the upper femur occurs early in prostatic malignancy.

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membranous bones and signs of diabetes insipidus (SEE p. 771).

Marble-Bone Disease (osteopetrosis): This is a condition in which the bones have undergone complete mineralization. The affected bones are whitish gray, are extremely brittle and show an



Fig. 13—Multiple congenital osteosis

entire absence of marrow space and of cortical demarcation

IV. **Tumors of the Bone:** The various bone tumors are classified as: (*a*) Benign and (*b*) malignant.

(*a*) ***Benign Tumors: Osteomata or Exostosis*** This usually occurs in the vicinity of the epiphyseal line of the long bones, the tumor being often covered by cartilage and capped by a bursa The two bones that are most frequently affected are the lower end of the femur and the ungual phalanx of the great toe.

An osteoma or exostosis is a bone tumor similar in structure to the bone from which it is an outgrowth, and occupies only a limited portion of its circumference, thus differing from hypertrophy, which involves the entire circumference of the affected bone. These tumors may be pedunculated or have a broad base. Their growth may be rapid or slow, usually painless and cause discomfort only because their presence may hinder motion or give rise to pressure symptoms

Ivory Exostosis: This is an osteoma; the bony growth is of great density and is found on the flat bones of the skull, in the orbit, in the auditory meatus, etc.



Fig. 14—Xanthoma tuberosum.
(Phila. Gen. Hosp.)

Xanthoma Tuberosum: This is characterized by the formation of nodules upon the extensor or flexor surfaces of the extremities. It is a connective tissue growth, usually multiple and found over

the joint and at other pressure points, *i. e.*, the knuckles, knees, elbows, palms, soles and buttocks. In these locations they often assume a bonelike hardness and may be mistaken for osteomata. Xanthomata occurring in other parts of the body, *i. e.*, neck, chest, mucous membrane of the mouth and the eyelids are

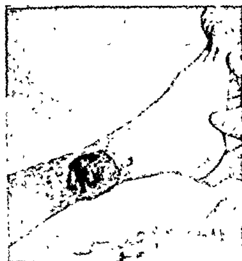


Fig. 15—Sarcoma of tibia. (Phila. Gen. Hosp.)

of softer consistency and occur in small nodular or flattened masses

Chondromata. These are cartilaginous formations that may occur upon the phalanges and the metacarpal bones, where they are usually multiple. Chondromata may develop upon any portion of the body which contains cartilage, therefore they are the most common of the benign tumors. The mass when superficial may be palpated as a hard though somewhat flexible tumor, it does not cause pain, but interferes with motion or causes friction because of its presence. A chondroma may undergo cystic degeneration and may at times grow in conjunction with a sarcoma or a fibroma.

Fibromata: These growths are likely to originate in the periosteum and most commonly affect the upper and lower jaws, though they may at times be found at the occiput, the vertebrae, the pelvis, the ribs, the sternum and the long bones. These fibrous tumors of bone are of slow growth, irregular in shape and of firm consistency. They do not cause pain, but may cause discomfort, because of their location, by pressure and by cystic degeneration.

Epulis: This is a fibrous tumor originating from the periosteum of the lower jaw and is sarcomatous in character.

Lipomata (very rare) may grow from the outer layer of the periosteum.

(b) *Malignant Tumors: Periosteal Sarcomata:* These are of various types and of differing degrees of malignancy. As a general rule, the softer they are in consistency and the more closely they resemble the embryonic type of tissue, the more malignant they are; small round-cell and spindle-cell sarcomata are more malignant than giant-cell sarcomata. Sarcoma may originate in a bone or may occur secondarily, as a metastasis from another viscus. A periosteal sarcoma is usually found at the end of a long bone—and as a rule grows rapidly; it causes little if any pain and always occurs in young individuals, and is accompanied by rapid loss of weight and strength.

This type of sarcoma often follows an injury. It is as a rule not very painful, and is associated with dilated veins over the tumor, and enlargement of the neighboring lymphatic glands. Metastasis occurs through the blood stream and most commonly affects the lungs, though the liver and other organs may be invaded. When metastasis takes place, it is evidenced by anemia, general weakness and

membranous bones and signs of diabetes insipidus (SEE: p. 771).

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will show areas of decalcification having a moth-eaten appearance. Bence-Jones albuminuria is usually present.

V. **Cysts:** Bone cysts may be classified into four types: (SEE: Fig. 8, p. 729.)

1. ***Cystic Degeneration of Bony Structures:***

(*a*) *Osteitis Fibrosa Cystica:* This is characterized by cyst formation of the ends of long bones, *i. e.*, the femur, humerus and tibia. They are usually painless and of long duration, often resulting in either breakdown of the cyst or fracture of the affected bone. This condition may follow traumatism, but is usually due to hyperparathyroidism (SEE. p. 789).

(*b*) *Dentigerous cysts* (follicular odontoma): These usually occur soon after the second dentition and are due to an excessive number of dental follicles. They appear as bony shells in the gums beneath the tooth margins, are crepitating to pressure and often contain one or more teeth.

(*c*) *Osteomalacia* (*Mollities ossium*) *and Osteoporosis:* These are characterized by softening of the bones, resulting in deformities of the limbs, spine, thorax and pelvis. They are associated with muscle pain, great weakness, anemia and other signs of a deficiency disease. There is a lack of calcium, phosphorus and other osteoid tissue. It is associated with steatorrhea and is often seen in pregnancy.

2. ***Degeneration of a Bone Tumor:*** In this type, the cyst is the result of cystic degeneration of a previously existing tumor, *i. e.*, giant cell sarcoma, myxoma, chondroma or a fibroma. These may be diagnosed because of the occurrence of softening over a previously hard mass.

3. ***Cysts Not of Bony Origin:*** These are hydatid and dermoid cysts; they are rare and when present may be recognized by their size, fluctuation, absence of pain and their benign tendencies Aspiration and examination of the cyst contents usually reveal their character



Fig. 18—Hydatid cysts in the deltoid muscle

4. ***Syphilis, congenital or acquired,*** may at times cause cystic degeneration of bony structures. The diagnosis may be suspected from the history and positive blood or spinal fluid findings

The Joints

Physical Examination of the Joints

The joints are examined for size, mobility and signs of inflammation.

Size: When only one joint is involved, its size should be compared, by actual measurement, to the corresponding joint on the other side. If similar joints on both sides are affected, then the relative size can only be judged by comparing them to the other joints of the body and to one's mental picture of a normal joint.

cachexia and such local symptoms as may be produced by the affected organ.

Endosteal or Myeloid Sarcomata: These are of very slow growth; they usually affect the ends of the long bones; *i. e.,* the lower end of the femur and upper end of the tibia; the upper end of the humerus and lower end of the radius, the sternal end of the clavicle, and the upper jaw. *This form of sarcoma is the* least malignant and seldom gives rise to metastasis or lymphatic enlargement. Pain over the mass occurs at an early stage of its development. The tumor is hard during the early stages and becomes softer as the outer shell of bone is broken through, yielding crepitation on pressure.

Carcinoma: This is always secondary to carcinoma elsewhere in the body. Thus carcinoma of the jaw may follow carcinoma of the lip or mouth. An epithelioma of the leg may cause a squamous celled carcinoma of the tibia. It is usually the spheroidal celled carcinoma which metastasizes to the bone, particularly from the breast or the thyroid gland *Carcinoma of a bone is usu*ally not very painful but causes spontaneous fracture of the affected bone.



Fig 16—Melanoma.

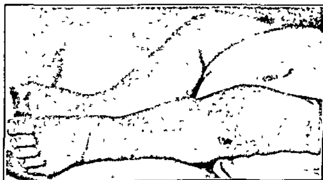


Fig 17—Sarcoma of knee.

Multiple Myeloma These are primary malignant neoplasms originating in the cancellous tissue of bone composed of bone marrow plasma cells They occur in the ribs, sternum, vertebrae, skull, clavicle, and ends of long bones. X-ray

Heberden's nodes: These are circumscribed swellings situated on the terminal phalanges of the fingers, frequently noted in rheumatoid arthritis and found in elderly subjects, apparently in perfect health.

Henoch's Purpura: This is often associated with acute swellings of the joints; it is as a rule found in children

Gout: This is characterized by swelling of the joints, particularly those of the large toe and thumb The swelling is usually due to so-called "chalk deposits" or sodium biurate crystals.

Scurvy: This is a deficiency disease due to lack of vitamin C Hemorrhages may occur subperiosteally and into the joints, causing the joints to become tense and swollen

Hemophilia, purpura and other blood dyscrasias may cause extravasation of blood in various joints and simulate arthritis.

II. **Chronic Joint Affections:** ***Osteoarthritis:*** *This is primarily a dis*ease of the cartilages and bones, causing a destruction of the cartilage with the formation of a bony joint, which gives rise to the formation of bony outgrowths or excrescences (*hypertrophic arthritis*).

Tuberculosis: This is more frequently found in children than in adults; usually one joint, especially the hip, is affected, although any joint or bone in the body may be the seat of this disease. The disease is slow in its progress, and causes suppuration with sinus formation associated with wasting of the muscles around the joint or affected part.

Syphilitic Arthritis: This has already been discussed under syphilis of the bones; it may be due either to acquired or congenital syphilis. The joint is usually enlarged, not very painful or tender to touch, and the diagnosis often depends upon other signs of syphilis



Fig 20—Charcot's elbow joint.

Charcot's Disease: This is associated with multiple cerebrospinal sclerosis and often with locomotor ataxia; it is characterized by great swelling of one or more joints which are sometimes associated with effusions. The knee, hip and elbow joints are most frequently affected Charcot's joint disease is usually recognized as occurring in the course of diseases of the spinal cord, as in tabes or syringomyelia, and leading to chronic synovitis affecting one or more joints, to brittleness of the bone, wasting of the articular extremities, and dislocation.

Palindromic Rheumatism: This is an acute recurring afebrile condition affecting a small or a large joint or its adjacent tissue, causing pain, redness, and swelling The lesions resemble angioneurotic edema. The attacks are sudden, disappear within a few hours or days and may reappear at various intervals.

Mobility: The affected joint should be gently manipulated so as to determine the degree of motion and the presence and absence of pain during motion.

Signs of Inflammation: An inflamed joint is usually swollen, red, tender to touch and painful in motion.

Joints are also examined as to fluctuation and for the presence of any other abnormality.



Fig. 19—Arthritis deformans.

Diseases of the Joints (Arthritis)

Joint affections are divided into two groups: *Acute and chronic,* which may or may not be followed by permanent joint deformities.

I. **Acute Joint Affections:** ***Acute Articular Rheumatism*** (acute rheumatic fever): The joint affected is red, swollen, tender to touch and very painful to motion. Several joints are usually affected at the same time, the infection often migrating from one joint to another. When recovery has taken place, the affected joints assume their normal size and shape There are no joint sequelae.

Septic Arthritis: As a rule, one joint only is affected. At times more than one joint may be affected, but the condition does not migrate to other joints, as in the case of acute articular rheumatism. Suppuration often occurs and permanent joint damage may result.

Pneumococcic Arthritis: This sometimes appears as a sequel to lobar pneumonia; it may affect any of the joints, as well as the middle ear.

Gonorrheal Arthritis: This usually attacks one joint, the knee being the most frequently affected; at times, particularly during the acute course of a gonorrheal infection, several joints may be simultaneously complicated. They are extremely painful and usually cause permanent damage

Arthritis Due to Other General Infections: This may also occur as a *complication or sequel* to typhoid or paratyphoid fever, scarlet fever, meningitis, influenza, measles, diphtheria and smallpox

Arthritis Due to Focal Infection: This is often overlooked or given a different name. One or more joints may be extremely painful to touch and motion, the swelling is not very large, and the discoloration of the skin is not very noticeable, the most prominent feature being an associated tenderness of the muscles near the joints. This form of arthritis may occur as a result of a focal infection in the tonsils, tooth sockets, gastrointestinal canal, prostate, anus, gallbladder, sinuses, or any portion of the body harboring a suppurative process

Acute Secondary Arthritis: This is an inflammation of a joint caused by an osteitis, as seen in osteomyelitis.

Rheumatoid Arthritis (arthritis deformans): This usually affects the small joints of the body and causes permanent deformity *(atrophic arthritis).*

and many other skin diseases are frequently found on the arms.

Scars: Most scars are a result of trauma. Among these may be included vaccination scars and those caused by careless hypodermic injections. Scars may assume various shapes and sizes depending upon the nature of the original cause. Certain skin diseases form ulcers which in turn cicatrize, *e.g.*, syphilis, leprosy, etc.

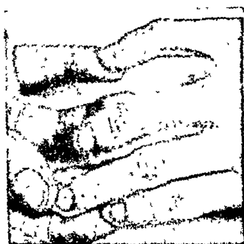


Fig 22—Hypogenesis of phalanges (Case of Dr Krusen)

Tumors: These may be either of the soft parts as myomata, lipomata, fibromata, neuromata, cysts; or of the hard structures, such as chondromata, sarcoma, or carcinoma.

Painful Areas: These may be due to neuritis, neuromata, osteomyelitis, tuberculosis and parectasis (excessive stretching or distention).

Anesthetic Areas: These may be due to spinal cord lesions and to leprosy.

Tenderness of the Joints: This may be caused by any form of arthritis, local infections, fractures, dislocations, Raynaud's disease, occupational neurosis, injuries of the soft parts and interference with the circulation or innervation.

The Hands and Fingers

Abnormalities of the hands and fingers may be congenital or acquired. The most common of these abnormalities are as follows:

Spadelike Hand: The hand is large, coarse and broad, the fingers thick and square, with broad nails, such as is often seen in myxedema. If bone as well as soft parts take part in the enlargement, deformity may be caused by acromegaly.

Claw Hand: This deformity usually occurs as a result of paralysis and atrophy of the interossei muscles, and is seen in amyotrophic lateral sclerosis, syringomyelia, and often in chronic anterior poliomyelitis and postencephalitis. The fingers and hand are contracted, resembling a bird's claw.



Fig 23—Polydactylism (supernumerary finger).

Hypogenesis of Phalanges: Several fingers are abnormally short in relation to one or two normal fingers, or one finger may be abnormally long, possessing an extra phalanx (congenital).

Supernumerary Fingers: These may occur as a congenital malformation.

Hypertrophic Pulmonary Osteoarthropathy: This is characterized by enlargement or clubbing and curving of the nails of the fingers and toes. Usually there is an associated enlargement of the wrist and interphalangeal joints. The lower end of the tibia and fibula may also be affected and occasionally there may be enlargement of the lower jaw.

This condition is frequently found in tuberculosis of the lungs, chronic bronchitis, bronchiectasis, chronic cardiac affections and in congenital heart disease

The Extremities

The Upper Extremities

The upper extremities are examined for nutrition, development, the presence or absence of pulsating vessels, the mobility of the joints, the condition of the fingers and fingernails, and the presence or absence of tremors

The Arms

The arms are examined for musculature, color, general nutrition and possible existence of tumors and painful areas

Color: The arms are usually of the same color as the rest of the body, except in persons who expose their arms to the sun, like farmers, longshoremen, sailors, hodcarriers, and foundrymen, or open-air bathers (sun or water).

Cyanosis of the arms is often seen in cases of *heart failure*; cyanosis of one arm may be caused by *local conditions constricting the venous circulation of* that member; *arteriovenous aneurysm* near the elbow joint may cause a like discoloration.

Redness is caused by acute inflammation and local irritation *Other colorations* may be due to staining by certain dyes or to constitutional diseases, *e.g.* jaundice, argyria, polycythemia, Addison's disease, etc

Rashes: Various skin diseases display their characteristic lesions upon the arm as well as upon other parts of the body *psioriasis* is most frequently noted on the extensor surfaces, particularly the elbows. Yellowish spots are often seen upon the arms of those who are *subject* to freckles elsewhere, and *eczema, pemphigus, granuloma fungoides, pellagra*

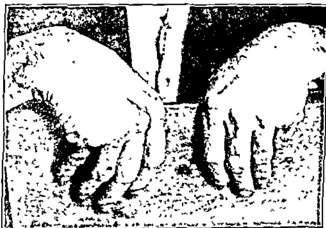


Fig. 21—Claw hand.

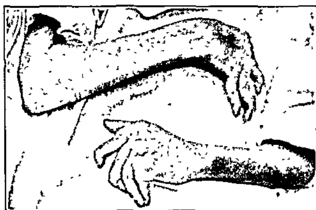


Fig 27—Rheumatoid arthritis.



Fig 28—Hemangioma.



Fig 29—Polyneuritic wrist drop.

Supernumerary fingers and toes are at times found in those presenting Laurence-Biedl's syndrome, and often in their close relatives who are otherwise well.

Clubbed Fingers: Decided clubbing is noted at the distal phalanges, accompanied with roughening of the nails (osteoarthropathy). This is often observed in chronic diseases of the lungs and heart; at times it is a congenital condition and is sometimes termed toxicogenic osteoperiostitis ossificans or Bamberger-Marie disease.



Fig. 24—Pulmonary osteoarthropathy (clubbed fingers).

Distorted Fingers: These are noted as a result of employment in certain occupations or of badly united fractures, or from the effects of arthritis deformans, and at times as a result of chronic rheumatism *Dupuytren's contraction* is a permanent flexion of one or more fingers arising from contraction of the palmar fascia and its digital prolongations.



Fig 25—Web fingers

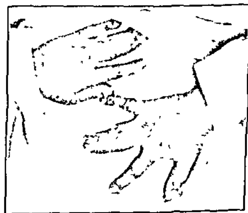


Fig. 26—Syndactylism hands.

Web Finger: As the name implies, the fingers are held together by a web of skin, not unlike the wing of a bat, or the foot of a duck or goose.

Abnormalities of the Nails: *Cyanosis* of the fingernails usually indicates poor circulation, anemia, and venous stasis.

Hard, brittle, and *longitudinally-grooved nails* are found in gouty individuals.

Dry, malformed nails may be caused by trophic changes, resulting from injury to the finger or nerve and are also noted

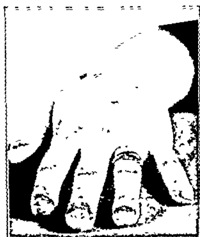


Fig 32—Acrodermatitis. (Raynaud's disease)

in neuritis, Raynaud's disease, pulmonary osteoarthropathy, syphilis, onychia, scleroderma, acrodermatitis and granuloma fungoides affecting the fingers

Ulcers and *ecchymosis* at the base of the nails if not due to trauma are often noted in chloral addicts or in syphilis and scrofula. A small indolent ulcer near the nail, especially if indurated and associated with enlarged lymph glands above the inner condyle, should arouse suspicion of a chancre. A small indolent ulcer near the nail accompanied by an enlarged axillary gland and fever should arouse suspicion of tularemia

Megalonychosis (Keyes) is an enlargement of the nail in its lateral dimensions not accompanied by defective structure; this may be a congenital condition

Quincke's capillary pulsation is a rhythmic flushing and blanching of the fingernails. This is seen most frequently in aortic regurgitation, but often, also, in anemia.

The Lower Extremities

The lower extremities are examined for color, condition of the skin, and condition of the musculature, bones, joints and vessels. Any deformities and painful areas should be noted and an attempt made to elicit both the normal and abnormal reflexes.

For the examination of the color and skin see p. 127 and for reflexes see p 831

Muscles

Atrophy of the muscles may be caused by disuse, either because of enforced rest or on account of disease of the brain, the spinal cord or of the nerve supply of the legs, fracture of one or more of the bones, or disease of the bones and joints. Atrophy of the anterior and outer muscles below the knee is seen in the peroneal type of progressive muscular atrophy.

Enlargement of the muscles of the legs, particularly of the calves, is noted in children suffering from hypertrophic muscular paralysis

Bones

The bones of the lower extremities may become affected similarly to the bones elsewhere. The following deformities are often encountered.

Curvature of the Bones of the Leg: This may be due to rachitis, osteitis deformans, mollities ossium (osteomalacia), and cretinism.

Coxa Vara and Coxa Valga: When the angle normally formed by the long axis of the shaft of the femur with the

Syndactylism: This is characterized by the joining of two or more fingers or toes.

Acromegaly: The hands are broad, the fingers thick, rounded and sausage-

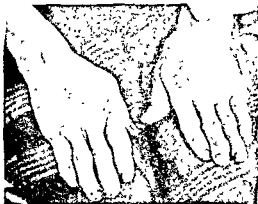


Fig. 30—Occupational deformity

like, and the fingernails are small. The bones are usually enlarged in proportion to the hypertrophy of the soft parts both as a result of abnormal deposits bony tissue in the joints and of parti dislocation of the affected parts.

Elephantiasis: This may affect o or more extremities or a greater part the body as a nonpitting edema.

Hemangioma: This is a rare cond tion. If an extremity is affected it ma attain an unusually large size.

Wrist Drop: This may result fro lead, alcohol, or arsenic intoxicatio disease of the spinal cord, and diseas or pressure of the brachial nerve, als from musculospiral paralysis, polyneu ritis, beriberi, diabetic neuritis and loca injuries In the author's ward at th Philadelphia General Hospital a man o 19 years of age developed wrist drop an ankle drop following acute gonorrhea

Occupational Deformities: Variou deformities occur as the result of occupation and should be differentiated from

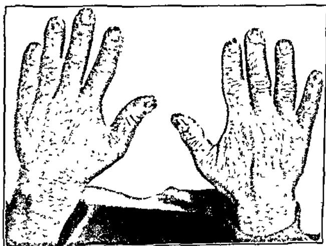


Fig 31—Granuloma fungoides.

Heberden's Nodes: These are knobby enlargements of the proximal ends of the terminal phalanges; this enlargement may be due to arthritis deformans and gout, but often its etiology is obscure

Rheumatoid Arthritis: This produces the most grotesque deformities, true arthritis. For example, the fingers of old washwomen, seamstresses and baseball players may resemble early cases of arthritis deformans To differentiate these conditions it is necessary to consider the history and to investigate other joints of the body.

Vessels[1]

Circulatory Disturbances: Visible arterial pulsations are caused by aortic regurgitation, or, if localized, by aneurysm.

Enlarged Veins of the Feet, Legs or Thighs: These are known as *varicose veins.* They are usually due to some interference with the return circulation of the lower extremities.

Increased Heat: This may be local or general Local increased heat may be caused by being in contact with a hot object, or as a result of local inflammation, and in erythromelalgia. General increased heat of the extremities is found in fever or when exposed to a heating object.



Fig. 35—Varicose veins.

Coldness: Local coldness may be due to interrupted arterial circulation and venous stasis. Coldness of one or both legs is found in Buerger's disease, arteriosclerosis, Raynaud's disease, and in pregangrenous states. General coldness may be due to diminished circulation and to exposure to cold.

Edema: This may be caused by heart disease, kidney disease, and certain anemias.

The Feet

Examination of the Feet: The *examination of the feet* is a matter of so great importance that it warrants a detailed description.

Nutt[2] recommends the following routine in examining the feet:

Inspection: This should begin with the patient's entrance into the examining room. Is there a limp? Is the foot held in abduction? Is the clothing over the internal malleolus worn? Are the inner ankles prominent? When the patient stands are the feet parallel or divergent? Are the soles flat on the ground, or do the toes turn upward? Are any of the joints, especially the first metatarsal-phalangeal, prominent through the shoe? Both feet and legs, above the knees, should always be bared for examination in every instance. First inspect the shoes; locate the most worn parts on the soles and heels: Is the upper stretched so as to overlap the sole or heel on either side? Is the inner side of the sole and heel on a straight line? Compare the height of the heel with that of the sole: Is the center of the heel under the weight-bearing part of the hindfoot? Then examine the stockings. Are they damp, are they pointed? Before their removal it had better be determined whether they constrict the toes. Note the color of the skin for signs of faulty blood supply. With the patient standing, notice the position of the toes: Are they

[1] See peripheral vascular disease, page 535

[2] Nutt: Diseases and Deformities of the Foot, E B Treat & Co.

long axis of its neck is considerably diminished, a condition known as *coxa vara* or "bent hip" results. If, on the contrary, this angle is abnormally increased, *coxa valga* (also called *collum valgum*), which is the more common condition, producing a marked external rotation, increased abduction and de-



Fig. 33—Genu varum (bowlegs in Paget's disease)

creased adduction results. *Coxa vara* may be either unilateral or bilateral. It is seen in growing bones, and most often in adolescents, because they are prone to undergo greater strains than young children. For the same reason males are more often affected than females When the affection is unilateral the left leg is more often affected than the right, possibly because more weight is thrown on this side in the "stand-at-ease" position. *Coxa valga* is really a widening of the angle made by the head and neck of the femur with the shaft, and is commonly mistaken for an early evidence of hip-joint disease. Its cardinal signs are abduction of the leg with external rotation and limitation of adduction.

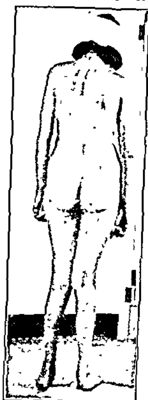


Fig 34—Genu valgum (knock-knee).

Genu Varum ("bowlegs"): This is a condition of the legs in which a line drawn from the head of the femur to the middle of the ankle falls inside the center of the knee joint (MacEwen) The knees are apart when the ankles touch, and the feet are often in a position of compensatory valgus.

Genu Valgum ("knock-knee"): This is the exact opposite of genu varum. It is an inward curvature of the knee or knees so that, when the legs are fully extended on the thighs an angle, salient internally, exists at the knee joints (Tubby).

Chronic, Painful, Hard Swelling of the Tibia: This may be due to syphilis or sarcoma.

ments to this tubercle. Pain about the external malleolus in cases of everted feet is due to a crowding of the tissues against the external malleolus from malposition of the tarsus, according to Golding-Bird. The pain about the inner side of the mediotarsal joint may be due to an inflammatory condition of this joint, or to strain.

Deformities of the Feet: Various deformities occur in the feet and toes, the commonest being *talipes* or *clubfoot.*

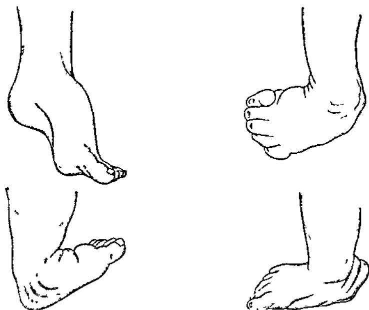


Fig 36—Various types of clubfoot

Clubfoot: The term *clubfoot* is defined by Tubby[1] as comprising those deformities in which the anatomical relations of the foot to the leg, or of one part of the foot to the other, are abnormal.

Inasmuch as the foot is capable of movements such as, dorsiflexion (flexion), and plantar-flexion (extension), adduction with inversion (supination), and adduction with eversion (pronation), talipes is associated either with overaction or loss of action of one or more groups of muscles affecting these movements. The following deformities may exist:

1. *Pes Equinus:* The heel is drawn up by contraction of the tendo Achillis so that the patient walks upon his toes, or in some cases upon the dorsum of the foot.

2. *Pes Calcaneus:* This is usually associated with pes valgus. The foot is drawn up to the leg, so that the patient walks upon the inner side of the heel This condition often follows infantile paralysis of the muscles of the tendo Achillis.

3 *Pes Varus.* Inversion of the foot causes the patient to walk upon its outer border, the sole being turned inward.

[1] Tubby. Deformities, Including Diseases of the Bones and Joints, 2nd Edit, vol 1, Macmillan & Co, London

flat on the ground, flexed, hyperextended, parallel? Is there a hallux valgus? Does the forefoot appear to be flattened out—extra wide? Is there a concavity or a bulging beneath the tuberosity of the scaphoid? Are the malleoli well defined? Does the outer one seem to be in its normal relation to the inner one, or is it apparently advanced? When examined from behind, do the tendinae Achillis run down vertically to the calcaneum or do they incline to one side? Are the normal depressions on either side the heel cord present? Does the heel spread out on all sides like an inverted mushroom? Ask the patient to rise on his toes: Is it easily done? Does the dome heighten? Are the ankles thrown upward and outward? Can the patient invert the feet and stand on the lower borders?

Palpation: Take one foot, the well foot first, if only one is complained of, on your knee in such a way that the entire leg is comfortable and relaxed The examiner's chair should be a few inches lower than the one upon which the patient is seated Note by feeling whether the local temperature is normal Search for evidences of uneven pressure, or of friction, such as calluses or corns If there are calluses under the forefoot, are they beneath each one of the five metatarsals, or beneath only the middle three? Is there callous formation along the outer border of the foot, or around the margin of the heel? Is there a bunion over the first metatarsal-phalangeal joint? Are there ingrowing toenails? Determine the condition of the circulation of the foot. If deformities of the toes are present, ascertain if they can be easily straightened by passive movements.

Hold the calcaneum firmly in one hand, with the tuberosity resting in the palm, grasp the bone with the thumb and fingers so as to prevent its moving, and with the other hand test the motion at the mediotarsal joint. Then hold the leg above the ankle with one hand and grasping the foot about the mediotarsal joint with the other, test inversion and eversion. Test the ankle joint last; in so doing do not let flexion and extension at the mediotarsal joint deceive you into attributing it to the ankle joint, so grasp the foot that the os calcis moves synchronously with the metatarsals Care must also be taken that the foot is moved in the vertical plane of the leg, otherwise abduction in dorsal flexion will exaggerate the true angle of flexion The range of active movements of all the joints, with the foot in resting position, should be determined

Pain is often of great significance in making a differential diagnosis, and the painful spots should always be definitely located Pain caused by pressure over a diseased or injured bone is usually more circumscribed and elicited more easily and definitely than the pain from pressure on a strained or ruptured muscle or ligament Stretching of a strained or ruptured muscle or ligament produces pain in a ligament only by separating the ends, but in a muscle, a contraction will produce it

Definite pain upon pressure over the body of the os calcis or of the first metatarsal is generally due to disease or injury to those bones Pronounced pain over the peroneal tubercle on the external surface of the os calcis, is due, according to Goldthwait, to a tension of the synovial sheath of the peroneal tendon dragging it away from its attach-

nutrition of the affected part, which may secondarily become infected with putrefactive microorganisms, resulting in either *dry* or *moist* gangrene The nutrition of a part may be interfered with by: (*a*) *Interference with the circulation* as in endarteritis obliterans, thrombosis, embolism, occlusion of a vessel by ligature, new growth, splints or tight bandage. (*b*) *Traumatism*, by bruising, crushing or exposure to intense heat, cold or chemical action. (*c*) *Disturbance*



Fig. 37—Gangrene of the toes

of innervation as in Raynaud's disease, erythromelalgia, peripheral neuritis, myelitis, syringomyelia and other lesions of the spinal cord. (*d*) *Constitutional disturbances* such as diabetes mellitus, leprosy, marasmus, cerebrospinal diseases and ergotism.

Moist gangrene usually occurs after a crushing injury or when dry gangrene becomes infected with putrefactive bacteria; it usually occurs at the distal part of an extremity. The affected part becomes extremely painful and is at first hot and red, later it becomes cold and bluish, and commences to slough. This is accompanied by a fetid odor of decaying animal matter. In favorable cases, a line of demarcation is formed which divides the diseased from the healthy portion of the extremity.

Dry gangrene results in mummifaction, the affected part becomes black, withers and often drops off. The part is cold and has no very offensive odor. Pain is often intense, particularly during the early stages. The line of demarcation between the gangrenous portion and the healthy part is usually an inflammatory zone.

Clavi (corns and callosities): These are painful, hard elevations of the skin usually occurring over the first metatarsal joints of the toes, most frequently on the small toes, often also upon the great toe or upon any of the other toes and upon the sole of the foot. They are usually caused by pressure.

Bunions: These are enlargements of the tarsal bones; the tissues covering them, because of pressure, become inflamed and painful; often a corn may develop upon its most prominent part

Gout: This is characterized by the formation of chalk deposits in the metatarsophalangeal articulation of the great toe, which becomes red, swollen and extremely painful



Fig 38—Gangrene (advanced).

Toenails: These may become hardened, thick and malformed, often interfering with the wearing of shoes.

4. *Pes Valgus:* The foot is everted so that the bones on the inner side of the knee and ankle are abnormally prominent; the arch of the foot is lost The patient walks on the inner border of the foot, the sole turned outward.

5. *Pes Cavus:* This form is subdivided by Tubby into *arcuarus* and *plantaris,* according to whether the front part of the foot is on the level with or below that of the heel, there being in each case a distinct increase in the convexity of the arch.

6. *Pes Planus:* This is undue flatness of the sole and arch of the foot, the arch being decreased or entirely wanting.

Compound deformities, such as talipes equinus and varus, or talipes calcaneus and valgus, are common.

Heredity: It has been observed that clubfoot runs in families Not only does clubfoot appear to be hereditary but the particular form reproduces itself in the offspring. With congenital clubfoot, other deformities, such as polydactylism, clubhand, harelip and spina bifida are frequently found.

Diagnosis: In dealing with talipes it is necessary to determine the type of deformity, then the cause. The method of examination is as follows:

1. The history.
2. The gait on entering the room.
3. The position of the foot and limb on standing and sitting.
4. An outline or impression of the sole of the foot.
5. General examination of the affected limb or limbs as to shape, size, muscular development, diminished or excessive mobility of joints, temperature of the limb; condition of the skin as to color, integrity and the presence of corns or thickened skin over the heels and beneath the balls of the toes.
6. The passive movements which may be effected by the surgeon, and the directions from which resistance is felt.
7. Localization of the resistant ligaments and fasciae, and of—
8. Contracted and paralyzed muscles. This is effected by touch, by movement on the part of the patient and by—
9. Electrical reactions of muscles.
10 Signs of abnormal and arrested development, especially of bones In congenital clubfoot, the presence of excessive inward rotation of the bones of the limb is a point of importance. Absence of the fibula or tibia, or parts of these bones, and a rudimentary patella, are occasional accompaniments. In paralytic equinovarus excessive prominence of the cuboid is an evidence of the duration of the affection.

Heel: Severe pain on pressure in one or both heels, particularly when walking, is often caused by periostitis of the os calcis. This condition may result from gonorrhea which may cause the formation of osteal spurs (gonorrheal heel). Heel pain may also be caused by arthritis other than of gonorrheal origin. X-ray examination may reveal an osteal spur, osteitis, or arthritis, but in some cases, it fails to reveal any pathology.

Toes

The toes, as well as the lower portion of the foot, may become abnormally *red* because of frostbites, or in the early stage of endarteritis obliterans (Buerger's disease), Raynaud's disease and erythromelalgia.

Black discoloration of the foot and toe indicates a gangrenous process.

Gangrene: Gangrene of the toes, feet or of any other portion of the body is primarily due to interference with the

pelvis and depressions of Scarpa's triangles. The diagnosis of hip joint dislocation should always be confirmed by x-ray examination.

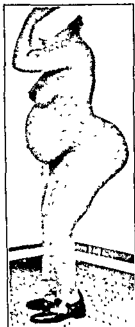


Fig 41—Pregnancy at term and congenital dislocation of both femurs

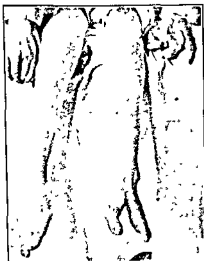


Fig 42—Deformity.

Knee: "Knock-knee" and "bowlegs" are described in this chapter (SEE p. 744)

Ankles: The various forms of clubfoot have been described (SEE: p. 747).



Fig 43—Polydactylism.

Feet: Various deformities as to shape and size of the feet have been noted The feet may be absent, a rudimentary knob surmounting the ankle or two flipperlike appendages displacing one or both feet. These deformities are usually due to an absence of one or more of the bones of the feet.

Toes: One or more toes may be absent One or more toes may be rudimentary. Web toes occur as frequently as web fingers, often in the same individual.

Supernumerary Toes (polydactylism). Supernumerary toes are a fairly frequent occurrence This condition usually runs in families Several members *of the same family may present this* anomaly. This condition is frequently found in Laurence-Biedl's syndrome (SEE: p 77)

Morton's Toe (anterior metatarsalgia): This is characterized by paroxysmal attacks of acute pain, usually in the fourth metatarsophalangeal articulation. It may also affect the second or third toe, but never the first It most frequently affects the right foot. The pain

is felt while walking or standing in shoes. Removing the shoe or manipulating the toe usually relieves the pain. Plantar flexion of the affected toe also causes pain Trauma and hereditary defect of the foot are believed to be etiologic factors.

Morvan Syndrome: This is a rare condition of unknown origin, first described by A. Morvan of France in 1883 It is characterized by the loss of pain and thermal sense in the hands and feet, retention of tactile sense, and painless ulceration with suppuration of the affected parts. The reported cases were in children of both sexes. Among the first symptoms are absence of pain from burns or other trauma This is followed by a general loss of pain and temperature sensitivity and occasionally of touch in the distal parts of the four extremities Later, trophic changes appear in the affected parts These are noted as changes in the nails, atrophy of bone, and ulcers in the soft tissues Accompanying infections may cause abscesses of the fingers, palms, or feet, requiring amputation Generally there is no weakness or paralysis of the muscles in other parts of the body, and no involvement of the upper neuron or of the lateral or the pyramidal tracts. This syndrome is to be differentiated from syringomyelia, leprosy, Raynaud's disease, and hypertrophic neuritis

Elephantiasis (Lymphedema)

This is a chronic disease due to obstruction of the lymphatic circulation It is characterized by enlargement of the affected part, which imparts a nonyielding "dead rubber" sensation to the palpating hand and does not pit on pressure. It may affect the extremities and the genitalia (SEE: pp. 546, 752, 1076, 1080).

Milroy's Disease: This is a familial type of lymphedema where several members of the family are affected.

Parasitic Elephantiasis: This is usually caused by filarial infection. The parasites may obstruct the lymph channels or they may form abscesses along the lymphatic course.

Sporadic or Idiopathic Elephantiasis: This occasionally affects young girls. One lower extremity and at times the genitalia may be affected

Panniculitis

This is a chronic inflammation of the panniculus adiposus It is commoner among women than men The affected areas have a hard brawny feel and are tender to manipulation The lesions occur subcutaneously over the inner surfaces of the arms and thighs and over the abdomen and chest as small masses, usually the size of a pea Larger tender rounded masses may also occur at the lateral aspects of the knee and ankle joints

Weber-Christian's Disease or Relapsing Febrile Nodular Panniculitis: This is characterized by recurring attacks of fever and the formation of painful nodular inflammatory swellings in the subcutaneous fatty tissue The lesions may undergo necrosis causing atrophy and depressions of the skin

Diffuse Panniculitis: This form is characterized by the involvement of fairly large areas of the subcutaneous tissue of the deltoid regions, the back of the neck, large areas of the back or elsewhere. The skin and subcutaneous tissue over the affected areas are thickened and tender, as is seen in adiposis dolorosa (SEE p 770)

CHAPTER XXVI

Anatomy, Physiology and Diseases of the Endocrine System

The endocrine system is composed of the following glands: (1) The pituitary, (2) the thyroid; (3) the parathyroids, (4) the adrenals; (5) the gonads (ovaries and testes); (6) the islands of Langerhans; (7) the thymus; and (8) the pineal.

The carotid body, the spleen and several other glands, while suspected of possessing internal secretions, are so far not generally included in the endocrine chain. On the other hand, the thymus and pineal glands, though not proven to possess specific hormones, are nonetheless included in the endocrine system This is done because they, like the other endocrine glands, exert a definite influence upon the development and maturation of the fetus and the infant

The Greek term "endocrine," or its derivative, endocrinology, was generally adopted after Claude Bernard in 1855 spoke about the presence of an "internal secretion" (ἐνδον — within, and κρίνειν—to separate) in the glands which Haller, in the 18th century, called "ductless glands"

Physiology: The function of the endocrine system as a whole may be summed up as being that of self-preservation and the preservation of the species These primary instincts are attributable to the combined actions of all the glands of the endocrine system which, because of their hormones, influence physical, mental and sexual development and reproduction.

Each of the ductless glands, by virtue of its hormone or hormones, is a specialized gland which plays a definite rôle, yet their individual functions are so interrelated that a defect in one gland may affect several other glands. Dysfunction of any one gland will cause a definite type of endocrinopathy. The type of endocrinopathy depends not only upon which of the glands has originally become affected, but also upon the severity of the affection, the kind of dysfunction and the extent to which the other endocrine glands have become involved

The Hormones: The internal secretion of an endocrine gland is known as a "hormone" (from the Greek ὁρμάνειν, to excite or arouse) This term was applied to it by Starling in 1905 and has since come into general use. The hormones are chemical substances possessing definite formulae. Several of the hormones are now being reproduced synthetically in the laboratory.

Each hormone, as it is absorbed by the circulation coursing through the gland in which it is produced, exerts a definite chemical or physiologic action upon the body An increase or diminution in the amount of secretion as required by the body results in either a hyper- or hypoactivity of certain functions of the individual. The quantity of hormone produced by each gland may depend upon the condition of the individual gland, the condition of the pituitary gland which influences that particular gland, the reciprocal action of other endocrine glands and the bodily requirements

The action of the hormones also depends upon several factors: (a) The

The Pituitary Gland

Anatomy and Physiology of the Pituitary Gland

The pituitary gland is the most important of the endocrine glands. It bears that distinction because of its many hormones which have reciprocal action with nearly all the other glands in that system.

Anatomy: The pituitary gland is a small somewhat elliptoid reddish gray body. In the adult it weighs between 0.6 and 0.8 Gm and is somewhat larger in the female than in the male. *It is situated within the sella turcica, being* suspended from the floor of the third ventricle by the infundibulum, which is in close contact with the hypothalamus. A tough membrane formed by a circular fold of the dura mater, the diaphragm sellae, covers the sella and its encased pituitary gland, leaving only an aperture for the passage of the infundibulum. The size of the normal sella turcica is approximately 13 by 16 mm.

The pituitary gland is composed of four lobes or structures

(a) *The anterior lobe* or pars anterior *is the largest lobe and is made up of various types of epithelial cells.*

(b) *The posterior lobe,* pars posterior or pars nervosa, is smaller than the anterior lobe and is partially surrounded by it; it is made up chiefly of a specialized type of glial tissue.

(c) *The middle lobe* or pars intermedia is a narrow strip lying between the anterior and posterior lobes; it consists of epithelial cells similar in structure, but not in function, to those found *in the anterior lobe*

(d) *The pars tuberalis* consists of a narrow *strip of epithelial cells* which covers the anterior *surface of the stalk* and is reflected on to the anterior part of the floor of the third ventricle.

Histology: The Anterior Lobe: *This is* composed of various types of epithelial cells which differ in their staining *ability, structure, size and function*

The chromophobes are the most numerous, *i. e*, about 52 per cent of the cells of the anterior pituitary, they contain *a nongranular cytoplasm and therefore do not stain readily by the ordinary* laboratory methods. Their function is not definitely known but it is believed that they are the mother cells or chief cells, held in an undifferentiated state, from which the other anterior pituitary cells are evolved according to specific requirements.

The chromophils make up the other 48 per cent of the cells, they contain granular cytoplasm and are *readily* stainable. The chromophils are of two types: One type, the eosinophils, acidophils or alpha cells, are stainable with acid stains such as eosin, hematoxylin and acid fuchsin; they constitute about 37 per cent of all anterior lobe cells. These cells elaborate the growth hormone as well as several other glandular *energizers. The other type, the basophils, basophilic cells or beta cells, are* the remaining *11* per cent of the cells belonging to the chromophil group; they are stainable only with basic dyes such as methylene blue, etc. These cells secrete the sex hormone as well as other energizing principles.

While the three types of cells just mentioned are the chief cellular constituents of the anterior pituitary body there are also new types of cells which make their appearance under certain

circumstances and at certain times. These are: (*a*) Cells of pregnancy, which develop in large numbers during gestation; (*b*) cells of castration, which make their appearance in the anterior pituitary body in castrates; and (*c*) *neutrophilic* cells, which increase in number with age. Whether these cells are new creations or are metamorphosed from preexisting cells is not known

The Posterior Lobe: This is made up of neuroglia cells which are identical with those found in other nervous tissue, of pituitocytes, which are highly branched cells containing a granular cytoplasm, and of nerve cells. The posterior pituitary body contains two hormones, pitocin, an oxytoxin, and pitressin, a vasopressor. There is some doubt as to the origin of these hormones. Some observers believe that pitocin and pitressin are elaborated in another structure, possibly the pars intermedia, and are stored in the posterior lobe; others believe that the posterior lobe actually secretes these hormones.

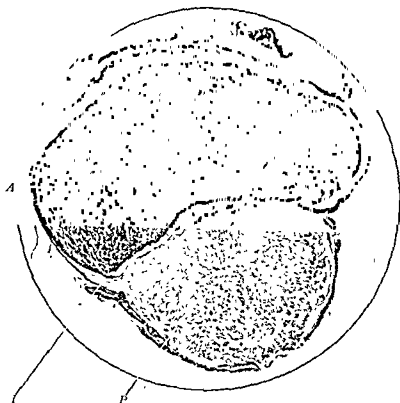


Fig. 1—Photomicrograph showing A, the anterior lobe, P, the posterior lobe and I, a remnant of the intermediate lobe The pars tuberalis is not shown (Courtesy, Dr H E Riggs, Philadelphia General Hospital)

The Pars Intermedia: This is composed of two types of cells: (*a*) Polygonal cells resembling the chromophobes, and (*b*) elongated threadlike cells stainable by the Golgi method This lobe elaborates a chromatophore *stimulant* known as intermedin, which also has water balance influence.

The Pars Tuberalis: This is made up of squamous cells and numerous vesicles; its function is not known. Some authors do not consider the pars tuberalis as being a fourth pituitary structure. They therefore hold that the pituitary gland is composed of only three lobes, namely, the anterior, posterior and intermediate lobes

The pituitary gland is of ectodermal origin; it receives its blood supply from the circle of Willis, the internal carotid and from the vessels of the stalk; its venous return is through the circle of Willis. The nerve supply is chiefly from the carotid plexus and sympathetic.

Pituitary Hormones

Seventeen substances have thus far been identified with the three lobes of the pituitary gland Several of these have proven to be of definite clinical value while others are still in the experimental stage These substances are generally alluded to as hormones. Each substance either has a definite effect upon the organism as a whole or energizes other endocrine glands to secrete their individual hormone in sufficient quantities

The anterior lobe secretes 14 hormones, the posterior lobe two hormones, the intermediate lobe one hormone, the pars tuberalis either does not secrete any hormone, or if it does the hormone has as yet not been discovered

The Anterior Lobe Hormones: (1) ***The Growth Hormone:*** This is derived from the eosinophilic cells. It promotes the growth of bone and soft structures; this hormone is abundant, and most active during childhood and before the sex hormone becomes very active.

(2) ***The Gonadotropic or Sex Hormone:*** This is secreted by the basophilic cells; it becomes abundant at puberty and continues its activity to the menopause. It is antagonistic to the growth hormone. The pituitary gonadotropic substance either consists of two hormones or one hormone that possesses two distinct principles:

(*a*) *Prolan A* is a follicle stimulating substance that acts upon the germ cells of both sexes It stimulates the granulosa of the ovarian follicle to ovulation and to the production of the ovarian follicular hormone.

(*b*) *Prolan B* acts upon the interstitial cells of the ovaries and testes. It luteinizes the theca cells, stimulates the production of true corpus luteum and the lutein hormone. This hormone is also responsible for the development of the secondary sex characteristics of the male and of the female

(3) ***The Thyrotropic Hormone:*** This stimulates the thyroid gland Ablation of the anterior pituitary causes thyroid atrophy and low basal metabolism This hormone is found in conjunction with other eosinophilic cell hormones

(4) ***The Adrenotropic Hormone:*** This stimulates the adrenal cortex and is found in conjunction with other basophilic cell hormones

(5) ***The Lactogenic Hormone*** (Prolactin and Galactin)· This promotes the secretion of milk after the mammary glands are prepared by the ovarian hormones Experimentally when the lactogenic hormone or hormones (there are probably two) are administered to properly prepared males or nonpregnant females they may be made to lactate.

(6) ***The Diabetogenic and Carbohydrate Metabolism Hormones:*** There are probably two principles: One

causes hyperglycemia and glycosuria by increasing the size and number of the islands of Langerhans; this hormone is called by some the pancreatropic hormone. The other is antagonistic to insulin; when it is administered to animals it causes hypoglycemia

(7) *The Fat Metabolism Hormones:* These are (*a*) the ketogenic hormone and (*b*) lipoitrin The ketogenic principle increases the ketone bodies in the blood; and lipoitrin, when used in small amounts, is said to cause an increased amount of fat to be stored in the liver, and when used in large amounts, it depletes the liver of its fat content

(8) *The Parathyroid Hormone: This increases parathyroid activity and* thereby raises the calcium content of the blood

(9) *The Nitrogen Metabolism Hormone:* This increases the specific dynamic activity during protein digestion

(10) *The Erythropoietic Hormone:* This stimulates the production of red corpuscles

(11) *A Bromic Hormone:* This was suggested by H. Zondek because he found stored in the anterior pituitary body large amounts of bromine which disappear from it during sleep

(12) *A Hepatogenic Hormone* This is said to influence the size of the liver and many of its functions

(13) *The Contrainsulin Hormone:* This is said to inhibit the action of insulin and to cause hyperglycemia and glycosuria

(14) *The Melanophoric Hormone:* This, principally, found chiefly in the intermediate lobe and also to some extent in the posterior lobe, is present in fairly large amounts in the anterior lobe Its action is that of influencing the chromatophores of cold blooded animals and probably has an effect upon pigmentation caused by diseases of the adrenal cortex.

Posterior Lobe Hormones: (1) *Pitocin:* This stimulates uterine contraction

(2) *Pitressin:* This raises blood pressure; contracts unstriped muscle fibers (excepting the uterus), is a respiratory stimulant, and has a diuretic and antidiuretic effect.

The Intermediate Lobe Hormone It is believed by some that the posterior pituitary lobe hormones are secreted by the intermediate lobe

Intermedin: This, a hormone directly *attributed to the intermediate lobe,* is composed of three principles (*a*) A phoxinus erythrophore-expanding principle; (*b*) a frog melaneophore-expanding principle, and (*c*) an antidiuretic principle effective in diabetes insipidus

Physiology of Pituitary Gland

Because of its many hormones or of a single complex hormone which influences the other glands of the body, the pituitary gland assists in governing nearly every function of the body An increased activity of the pituitary or of any of its energizing substances will result in a condition characterized by hyperactivity. The particular type of hyperactivity depends upon which of the hormones is secreted in excessive quantities. A diminution in any one of its secretions will result in hypoactivity of the particular function or functions affected by that specific secretion

Pathology

Lesions affecting the pituitary gland as a whole or any of its lobes or groups

of cells may be of various kinds. Those causing hypofunction are: (*a*) Atrophy of the gland as a whole or of any of its lobes because of vascular changes, pressure, or malnutrition; (*b*) destructive lesions such as certain types of tumors, cysts, abscess or aneurysm (SEE Fig 4, p 871); (*c*) constitutional diseases such as syphilis, tuberculosis, or other infections; (*d*) hereditary influence, and (*e*) reciprocal influence of other glands of internal secretion

Lesions causing hyperfunction of the pituitary are: (*a*) Hypertrophy or hyperplasia of the pituitary as a whole, or of any of its lobes or group of cells; (*b*) increased vascularity of the gland, (*c*) hereditary influence; (*d*) reciprocal activity of other endocrine glands, and (*e*) adenoma.

It is to be borne in mind that an adenoma because of its glandular structure causes hypersecretion and therefore hyperactivity, but when it becomes very large it may so compress the gland or some of its secreting cells as to interfere with function as may also other tumors or space-taking lesions which destroy or compress the gland The most common tumors are adenomata. and these may originate from any of the cell groups in the pituitary. Chromophobe adenomas grow to a very large size and compress the eosinophilic as well as the basophilic cells, thereby causing stunted growth and hypogenitalism. This type of tumor may outgrow the sella, destroy the clinoids, invade the cranial cavity and compress the optic chiasm, producing hemianopsia.

Eosinophilic adenomata are smaller than the chromophobe adenomata; often they stimulate the action of the eosinophils and cause gigantism or acromegaly. When a tumor causes destruction of the eosinophils during childhood, stunted growth is the result.

Basophilic adenomata are minute; often they are recognizable only on serial section. They are responsible for Cushing's syndrome. When the basophils are destroyed hypogonadism is produced.

Suprasellar tumors, when they compress the anterior pituitary, may cause in addition to intracranial pressure also pituitary symptoms.

Tumors affecting or compressing the posterior lobe or the stalk may cause diabetes insipidus

Diseases of Pituitary Origin

The type of pituitary endocrinopathies depends upon a number of factors: (*a*) Hyper- or hypoactivity of the gland as a whole, of any one of its lobes or of any particular group of cells within the lobe, (*b*) the time of life the affection developed, and (*c*) the concomitant affection of other glands Pituitary endocrinopathies are characterized by disturbances in the development of stature, of bones and of gonads; by changes in the distribution of fat and of hair; by the appearance of the skin, and by certain metabolic changes.

Hyperpituitarism

The gross characteristics of hyperpituitarism are those of hyperdevelopment of either the individual as a whole or of those parts or functions governed by a specific gland which in turn is stimulated by an overacting pituitary hormone The outstanding characteristics of hyperpituitarism are: Increased stature such as gigantism or acromegaly; increased hirsuitism; greater muscular development; increased vigor; hyper-

gonadism; and an increase in the various metabolic processes.

Diseases due to hyperpituitarism are: (1) Gigantism; (2) acromegaly, and (3) basophilism. These diseases, while primarily of hyperpituitary origin, also show evidence of other endocrine gland participation as the result of pituitary influence upon these glands

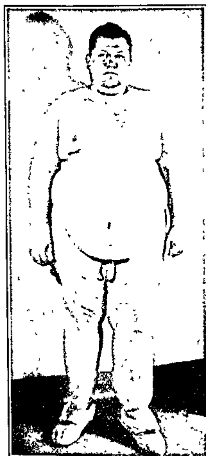


Fig 2—Pituitary gigantism, seven feet eight inches tall, weighing 414 pounds

1 Gigantism—Excessive Tallness (Preadolescent Hyperpituitarism): Gigantism is attributed to a hypersecretion of the growth hormone brought about by hyperstimulation of the eosinophilic cells of the anterior pituitary lobe by an adenoma, by excessive vascularity, or by irritation resulting from trauma or infection. Gigantism may originate during infancy, early childhood or during the adolescent period before the completion of epiphyseal ossification Ossification in these cases is delayed so that the individual may continue to grow in height well into the third decade.

The General Characteristics: There is skeletal overgrowth especially of the long bones, therefore all giants are abnormally tall. Because of individual peculiarities gigantism is loosely divided into five types (*a*) In uncomplicated macrosomia or simple gigantism the individual is very tall, and proportionately symmetrical in stature, extremities and viscera During the early stages there is increased vigor, hypertrichosis and often hypergonadism Later these give way to weakness, hypotrichosis and hypogonadism. (*b*) Pituitary gigantism is characterized, during the early stages, by general body overgrowth, with a tendency to an increase of the upper measurements over the lower, later girdle obesity and hypotrichosis may develop (*c*) Polyglandular gigantism starts very early in life; the individual grows rapidly, is generally thin and may develop diabetes mellitus, pulmonary tuberculosis, diabetes insipidus, and show evidence of other glandular defects (*d*) Eunuchoid gigantism is characterized by the excessive length of the extremities, poorly developed genitalia, female hair distribution in the male, long narrow face, long fingers and toes, and by easy fatigability (*e*) Acromegalic gigantism generally originates during adolescence when epiphyseal ossification is nearly completed; therefore these individuals, in addition to

gigantism, also develop some acromegalic characteristics. They usually show a massive lower jaw, a large nose, disproportionately large hands and feet. They have heterosexual hair distribution, are tall and seldom develop kyphosis.

Symptoms: Among the symptoms common to all types of gigantism, particularly during the later stages, are headache, hyperglycemia, cerebral pressure symptoms, asthenia and sexual hypofunction. The delayed epiphyseal union may be explained by the observation that the growth hormone is antagonistic to the sex hormone, and deficient sex hormone retards epiphyseal union

*2. **Acromegaly*** (Postadolescent hyperpituitarism): Acromegaly, like gigantism, is due to a lesion in the anterior pituitary lobe which stimulates the eosinophilic cells to an increased production of the growth hormone. Unlike gigantism the pathology becomes manifest *after* epiphyseal ossification has taken place so that skeletal growth is *not possible, and only such parts of the body become enlarged which are not influenced by the epiphyseal ossification*

The onset is between the ages of 20 and 40 years, and is of slow *progression*. It occurs in both sexes There is *no* elongation of the skeleton, the enlargement *is of the acral or peaked portions* of the body and of some of the viscera In a well-developed case the face appears massive, the nose is large; the supraorbital ridges and zygomae are prominent, the lower jaw is pugnacious, and the lower lip is prominent The teeth are widely spaced and the tongue is large. The neck appears short because of the upper dorsal kyphosis, the massive clavicles and the massive and prominent sternum. The hands are *large and spadelike* and all or occa-



Fig 3—Acromegaly, age 22 years, due to pituitary cystadenoma (Note acromegalic face and hands) (Courtesy, Dr N W Winkelman)

sionally only a few of the fingers are thick and sausage shaped. The feet and ankles are massive. The skin is often thick and furrowed. During the early stages there is hypertrichosis and hypergenitalism. X-ray examination will reveal epiphyseal tufting, irregular thickening of some of the cranial bones,

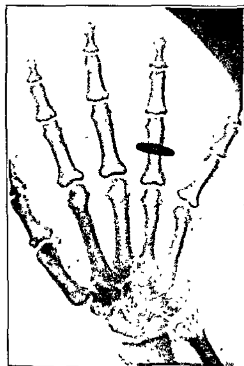


Fig 3a—Acromegalic hands (Courtesy, Dr. Leon Solis-Cohen)

and deepened grooves in most of the bones of the body in which lie tendons, blood vessels and nerves. The sella turcica may become enlarged, or the floor, the anterior or posterior clinoids may become eroded by a large tumor or an aneurysm. In the absence of such lesions the sella turcica will show no changes in size or contour

Symptoms: The most frequent complaints are pain in the bones and joints, headache, dizziness and digestive disturbances. Glycosuria, polyuria, and nephritic symptoms are fairly common. In the later stages, asthenia, hypogonadism, hypotrichosis and obesity are prevalent Prognosis as to life is generally favorable.

3. **Pituitary Basophilism** (Cushing's Syndrome): This condition develops in the presence of a basophilic adenoma which is often of microscopic size, or as the result of hyperbasophilism, the latter being characterized by hyalinization of the basophils Other glands, such as the adrenal cortex, the ovaries, the thymus, the thyroid, the parathyroid and the islands of Langerhans, also show evidence of pathology

This condition is more prevalent among young females than males, and particularly in those possessing a lymphatic hyperplasia The general characteristics are. Plethoric obesity, often painful, affects the face, shoulders, trunk and abdomen (girdle obesity); the upper and particularly the lower extremities are thin. Purplish striae develop over the breasts, lower abdomen and upper thighs During the early stages there is precocious sex development which later gives way to frigidity and sterility Heterosexual hair distribution with hypertrichosis in the female, and hypotrichosis in the male is quite characteristic. Osteoporosis, glycosuria, hyperglycemia and hypertension are fairly early manifestations Extreme weakness, backache, and headache continue to the last Cutis marmorata (transient mottling of the skin) of the extremities is common

Hypopituitarism

Endocrinopathies resulting from hypopituitarism vary with the structures affected and the time in the individual's

life that the affection began When the growth hormone alone is affected during childhood, growth remains arrested; if the structures governing both the growth and the sex hormone become affected during childhood, there results infantilism characterized by stunted producing cells become affected during adulthood, sex function stops and there develops a tendency towards hetero-sexual inversion. Other pituitary hypofunction may be manifested as obesity, cachexia, and various other structural and functional anomalies

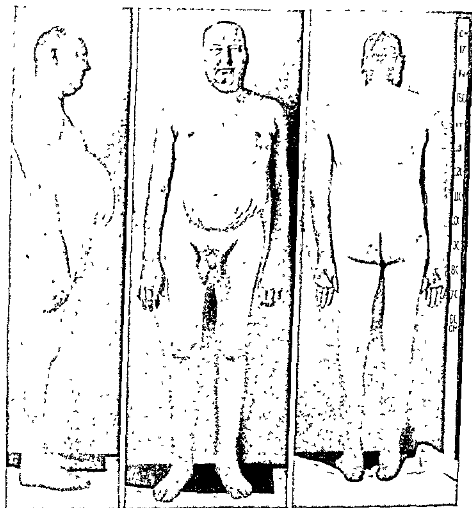


Fig 4—Lateral view Anterior view Posterior view Pituitary basophilism (Cushing's syndrome) ("Pituitary Body, Hypothalamus," Harvey Cushing, Charles C Thomas, Springfield, Ill)

growth and failure of sexual development Should the sex hormone cells alone become affected during childhood, then stature is unaffected as the child grows, but the sexual organs remain infantile; and when the sex hormone-

General Characteristics of Hypopituitarism: With few exceptions, hypopituitarism presents the following characteristics. The *skin is soft; there* is a sparse growth of body hair except *upon the head; in those old enough,*

the hair upon the mons veneris is of heterosexual distribution. The wrists and forearms, the ankles and legs are trim and small in proportion to the general development. In the presence of adiposity, the fat distribution is characteristic, being most pronounced in varying degrees in most cases. In the nonobese, and often in the obese, the upper measurement is greater than the lower.

Diseases due to hypopituitarism are (1) Infantilism and dwarfism; (2) Fröhlich's syndrome; (3) adiposis do-

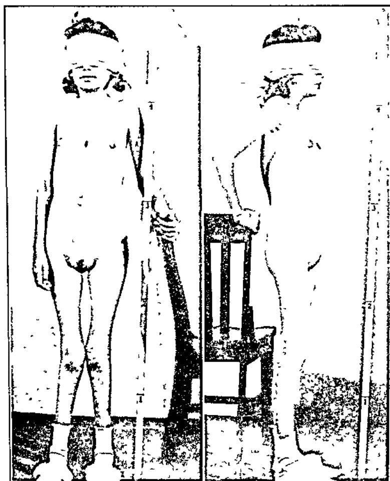


Fig 5—Infantilism. Age 18½ years

over the buttocks, hips and abdomen (girdle type of obesity) The basal metabolic rate is subnormal; specific dynamic action of protein is low; cholestrole is generally above normal, and carbohydrate tolerance is high. Sexual hypodevelopment or hypofunction occurs

lorosa; (4) Laurence-Biedl syndrome, (5) lipodystrophia progressiva; (6) pituitary cachexia; (7) diabetes insipidus; (8) Hand-Schuller-Christian's disease; (9) obesity, (10) pituitary headache; (11) pituitary somnolence and hibernation; (12) pituitary epilepsy, and (13)

abnormal hair *distribution of pituitary* origin.

1. Infantilism and Dwarfism: In this condition the individual remains infantile or dwarfed throughout life. The height of these individuals vary, depending upon how early in life the growth had become arrested or slowed. The genitalia are often in proportion to the size of the individual, as is also their function. Secondary sex characteristics are, as a rule, poorly developed, though occasionally gonadal function and secondary sex characteristics are present in a mild degree. The degree of development depends not only upon the time of life that the affection began, but also upon some inherited or congenital defect and upon the concomitant participation of other glands.

Types of Infantilism and Dwarfism: (*a*) *Pituitary Infantilism* (Lorain-Levy type): The individual is of child-like appearance with soft skin, round chubby face, round eyes and pouting mouth. All the features are proportionate, except that the trunk is somewhat longer than the lower extremities. The ossification centers are delayed. The mentality is average. The general appearance is that of an adult in miniature. The gonads are in proportion to the size of the individual, showing a general arrested development, both somatic and sexual.

(*b*) *The Thyroid Pituitary Type:* This type should not be mistaken for cretinism which it vaguely resembles. The stature is short; the features are coarse; the head is rounded, the skin is somewhat dry and harsh; the abdomen is enlarged, there is often lumbar lordosis; the limbs are large and round; the mentality is as a rule poorly developed. Before these individuals reach their thirtieth year they have wrinkled faces and look like little old men or women. The genitalia and the secondary sex characteristics are generally poorly developed. This type is also known as Brissaud's type of infantilism.



Fig. 6—Infantilism of Brissaud's type. Both children are of the same age. (Engelbach's "Endocrine Medicine," Charles C Thomas, Springfield, Ill.)

(*c*) *The Pituitary Gonadal Type:* This type of infantilism is associated with hypogonadism. In the male there may be cryptorchism and large breasts. In the female the breasts are rudimentary, and there is amenorrhea. In both sexes there is heterosexual hair distribution. The stature is generally below normal, though not as marked as in

the Lorain-Levy and the Brissaud's types. The individuals may be very thin or quite stout. The stout show the pituitary type of fat distribution and trochanteric fat pads. The lower measurements and the span are shorter than the trunk or upper measurement. This type should not be confused with the primary gonad type, which present very long legs and arms and a short trunk

(*d*) *The Thymicpituitary Type* This resembles the gonad type Here, too, the lower measurements and the span are greater than the trunk or upper measurements, though the genitalia are better developed Lanugo remains upon the body for quite an extended period The permanent teeth are bluish white, of poor architecture and disintegrate quite early; the second upper incisors and canines are rudimentary. The head is small and is well covered with hair and the general appearance is delicate In the obese type there is an associated lymphatic hyperplasia. The thin type presents long fingers and toes and a cylindrical type of body with a juvenile face.

(*e*) *The Adrenalpituitary Type* This type of infantilism is associated with premature puberty The trunk is longer than the lower extremities; pubic hairs appear quite early It may often be associated with either macrogenitosomia precox or with pseudohermaphroditism.

The Lilliputian. While resembling the Lorain-Levy type, this type has not been proven to be of pituitary origin These individuals, while of minute stature, often have quite normally functioning genitalia Several such dwarfs who married have been reported to have had children.

The Australian Pygmies While dwarfed, these people do not show any evidence of pituitary hypofunction Other dwarfs such as cretins, achondroplastics and mongolian idiots are believed not to be of pituitary origin.

2. **Frohlich's Syndrome** (Dystrophia Adiposagenitalis, Hypophyseal Dystrophy): This condition is fairly common; it may occur at any age and in varying degrees of severity It is characterized chiefly by adiposity and genital hypoplasia. The individual is usually fat, presenting the typical girdle obesity; the fat is distributed over the breasts, upper arms and thighs, and over the abdomen, mons and buttocks,

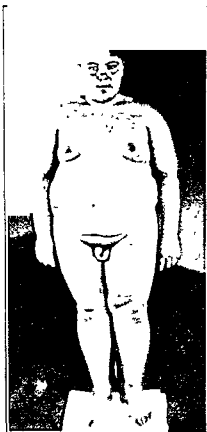


Fig 7 — Fröhlich's syndrome. Eleven years old. (Courtesy, Dr Michael Burns, Philadelphia General Hospital.)

yellowish discoloration of the skin; and xanthomata. This disease appears chiefly during childhood and more often among boys. In many, though not in all cases, there was found a pituitary lesion The symptoms are thirst, obesity of the pituitary type *with stunted growth*, and exophthalmus. The exophthalmus develops gradually, as the eye sockets become filled with xanthomatous masses and push one or both eyes forward Foam cells are found in the infiltrating tissue (SEE: Figs. 10 and 11, pp. 730 and 731)

9. **Obesity** (SEE p 88) This condition, irrespective of its etiology, is characterized by abnormal deposits of fat in various parts of the body or in all tissues where fat is depositable In all types of obesity there exists some disturbance in *the metabolic processes and a disproportion* between the intake of food and the output of energy. The concentration of fat deposits upon various parts of the body are not uniform in all obese persons For this reason obesity is at times classified into various types.

Pituitary Obesity is characterized by the large accumulation of fat over the *breasts and particularly over the buttocks* and abdomen, so that the abdomen hangs down apronlike, often in midabdomen there is a longitudinal constriction dividing the abdomen buttock-like into two lateral halves The ankles, wrists and forearms are thin

Hypothyroid Obesity presents a uniform distribution of fat with excessive *supraclavicular and supraspinous* padding. The breasts are large and the thighs and legs are massive. The skin is dry, inelastic and often leathery.

Hypogonad Obesity: While the individual may be fat all over, the greatest accumulation of fat is over the trochanters.

Adrenal Obesity resembles basophilism; the fat is distributed over the shoulders, upper arms and chest, the lower extremities are thin (Buffalo type of obesity).

Pineal Obesity is commoner in young boys and is associated with plethoric



Fig 10 — Lipodystrophia progressiva. Note the size of the lower abdomen and the lower extremities. (Philadelphia General Hospital.)

coloration, increased muscular development, and hypergenitalism (macrogenitosomia precox).

Other types of obesity are of cerebral origin as seen in cerebral tumors, salt and water retention and in other conditions.

10. **Pituitary Headache** (SEE: p. 68): Headache is a common symptom in many diseases. The pituitary gland is held responsible for a goodly number of headaches, particularly in women. Such headaches are found during menstruation, pregnancy, the menopause and often after castration, *i. e*, in conditions in which the pituitary enlarges or develops specific types of cells In acromegaly, obesity, Frohlich's syndrome and various pituitary tumors, headache is a frequent complaint The diagnosis is often made only by exclusion

11. **Pituitary Somnolence and Hibernation**: Certain types of hypopituitarism, such as obesity, cachexia, and destructive lesions of the pituitary, are accompanied either by transient uncontrollable attacks of somnolence or by prolonged comatose sleep from which the patient is aroused with difficulty. This condition is often seen in tumor of the pituitary and occasionally in the very obese who, though of the hypopituitary type, do not show evidence of tumor

12. **Pituitary Epilepsy**: Attacks of petit mal and occasionally of grand mal may be found in the pituitary type of young girls preceding puberty. These attacks often disappear when menstruation is well established. Tumor of the pituitary and intracranial crowding is a common cause for epileptic attacks. The so-called idiopathic epilepsy may occasionally have a pituitary background

13. **Hair Distribution of Pituitary Origin**: Several of the endocrine glands seem to participate in the growth and distribution of hair The gonads and the suprarenals seem to be the most prominent However, the pituitary gland which governs both the gonads and suprarenals as well as other endocrines seem to have a special trichogenous function. In hypopituitarism there is a heterosexual hair distribution, *i e*, an increase of body hair in the female and scanty facial and body hair in the male In acromegaly and in pituitary basophilism, hypertrichosis is the rule On the other hand, alopecia, congenital and acquired, has been found in several instances to be due to pituitary tumor

The Thyroid Gland

Anatomy and Physiology of the Thyroid Gland

The thyroid gland is composed of two lobes and a connecting narrow isthmus It weighs between 30 and 40 Gm and is located in the anterior portion of the neck below the cricoid cartilage, extending laterally beyond the anterior belly of the sternocleidomastoid muscle on each side. It is composed of a number of lobules lined with epithelial cells and contains a colloid material.

The hormone secreted by the thyroid gland is known as "thyroxin." The daily requirement of thyroxin to keep an individual's basal metabolic rate at a normal level is 0 75 mg. The normal basal metabolic rate is considered to be between minus 15 and plus 15. One mg of thyroxin will cause a 2 5 to 3 per cent increase in the basal metabolic rate. The physiological action of the thyroid is twofold· (1) In children it promotes body growth and bone development, the development of the nervous system and genitals, sharing these functions with the pituitary, the thymus, the suprarenal cortex and the gonads; (2) in the adult

it regulates metabolism, that is, the physiochemical processes of all tissues.

Disease of Thyroid Origin

Disease of the thyroid gland may cause *hypersecretion*, *hyposecretion*, or *perverted secretion* of its hormone, which may result in accelerated metabolism, decreased metabolism or perverted metabolism.

The thyroid gland may be the seat of various tumors, diseases, regenerative and degenerative processes; and it may become enlarged or atrophied with or without any secretory changes. It exerts a definite influence on body growth and metabolism, and though controlled by the thyrotropic hormone of the pituitary, it is one of the stabilators of endocrine balance.

Hyperthyroidism causes an exaggeration of all functions plus autonomic imbalance, *i. e.*, the heart becomes rapid, the mind is alert, often causing psychic disturbances, restlessness, excitement, tremors, hypertension followed by hypotension, mononucleosis, increased elimination of solids, diarrhea, hyperhidrosis, loss of weight and increased basal metabolism. Hyperthyroidism is of three types, *i. e.*, simple hyperthyroidism, toxic adenoma and exophthalmic goiter.

Hypothyroidism causes sluggishness of all functions. The patient is usually stout, though not invariably so; the mind is dull and muscular activity is



Fig. 11—Vascular supply of the thyroid gland, (semidiagrammatic). (*a*) Superior thyroid artery; (branch; (*d*) anterolateral branch; (*e*) inferior thyroid left recurrent laryngeal nerve. (Eberts, Surgical Disea

depressed. The degree of hypothyroidism in the adult governs the severity of the myxedema, and in the infant the degree of cretinism.

Athyroidism in the very young results in extreme degrees of cretinism; and in the adult in cachexia strumipriva or a severe type of myxedema

Dysthyroidism produces a perverted secretion which, according to Janney and Plummer, is responsible for exophthalmic goiter.

Enlargement of the Thyroid Gland

Any enlargement of the thyroid gland is classified as goiter or struma

Enlargement of the thyroid gland may be divided into three groups:

I **Thyroiditis:** Inflammation of the thyroid may be classified as acute nonspecific inflammatory, acute suppurative inflammatory thyroiditis and subacute and chronic thyroiditis. These may be due to local or systemic infection. Riedel's struma and Chaga's disease are special types of thyroiditis The symptoms, pain, redness, and swelling over the thyroid, are acute The pain often radiates to the teeth, occiput and shoulders The head is held rigid, the veins of the neck are prominent, and there is cyanosis of the face and neck. Swallowing and respiration, because of pressure, become difficult. Suppuration of the thyroid, when not fatal, may result in myxedema.

II. **Tumors of the Thyroid:** These may be of the following types: Carcinoma; sarcoma, malignant or simple adenomata; gumma, tuberculosis, syphilis and actinomycosis The benign tumors usually give rise to pressure symptoms only; the malignant tumors may cause pressure symptoms with signs of either hyperthyroidism or myxedema with cachexia. The internal secretion of the thyroid gland is often disturbed in such cases.

III. **Goiter:** This is an enlargement of the thyroid gland with definite changes in its structure. The following are to be considered: (*a*) Simple or vascular goiter; (*b*) colloid goiter; (*c*) parenchymatous goiter; (*d*) endemic goiter; (*e*) adenomatous goiter; (*f*) exophthalmic goiter (hyperthyroidism).

(*a*) ***Simple or Vascular Goiter:*** This is usually seen in young people, most often in girls at puberty and in young women during pregnancy and lactation. The thyroid is only moderately enlarged, is soft, free from pain and may cause symptoms of varying degrees of hyperthyroidism, *i. e*, hyperexcitability, elevated basal metabolic rate, sweating and tachycardia. The enlarged thyroids often seen associated with pulmonary tuberculosis or other conditions of the lungs which cause vascular stasis may be grouped under this heading.

(*b*) ***Colloid Goiter:*** This is simple nontoxic enlargement of the thyroid gland, at times it may attain to an enormous size and may give rise to pressure symptoms, or it may undergo degenerative changes producing cysts, calcareous infiltration, malignant changes or proliferative changes which may result in hyper- or hypothyroidism or cretinism

(*c*) ***Parenchymatous Goiter:*** This is a true hypertrophy of the gland In the chronic form the thyroid becomes quite large and fibrotic and there develop within its structure simple and colloid adenomata Ultimately the secretory function of the thyroid becomes impaired and hypothyroidism results Pregnant mothers suffering from paren-

chymatous goiters may give birth to goiterous offsprings that may be cretins.

(*d*) ***Endemic Goiters:*** These occur in large numbers in certain localities in Asia, Central Europe and in this country in regions far removed from the sea. fuse colloid goiter which may cause hyperthyroidism, hypothyroidism or may eventually involute.

(*e*) ***Adenomatous Goiter:*** This is usually seen in two stages: (1) Nontoxic adenoma, and (2) toxic adenoma.



Fig 12--Toxic adenomatous goiter The B M R was plus 36. (Philadelphia General Hospital)

There are two types One type is the diffuse parenchymatous colloid-poor goiter of childhood; and the other, the nodular adenoparenchymatous goiter with degenerative changes of the adult. The parenchymatous degenerative goiters of both childhood and adulthood are found in endemic cretins and severe myxedema. The other type is the dif-

1. ***Nontoxic adenoma*** may be single or multiple and usually occurs in the second decade of life. The mass or masses are generally circumscribed and firm to the touch Histologically they are made up of numerous acini, and occasionally of numerous circumscribed and encapsulated nodules containing many small alveoli. Colloid and cystic formations are

often found in conjunction with adenomatous tissue.

Symptoms: This form presents no definite symptoms or signs unless it becomes so large that it may cause pressure symptoms, or when it becomes toxic.

2. ***Toxic adenoma*** may be recognized as a hard circumscribed mass in one or both lobes of the thyroid associated with symptoms of hyperthyroidism. Often a nontoxic goiter may, because of overaction, cause toxic symptoms. These differ in their manifestations from true exophthalmic goiter in that the former contains an excess of normal thyroid secretion (thyroxin) while in the latter there is an excess of a perverted thyroid secretion causing severe toxic symptoms and requiring an iodine molecule for its readjustment (Plummer).

Symptoms: The onset may be gradual or abrupt. The gradual onset is manifested by increasing irritability, frequent attacks of tachycardia, weakness, digestive disturbances and functional nervous manifestations. A well-developed case will present the following: (*a*) Enlarged thyroid gland containing one or more hard nodes; (*b*) tachycardia, (*c*) coarse tremors of the hands and fingers; (*d*) nervous instability, (*e*) loss of weight [illegible] (*f*) myocardial degenera- [illegible]

If the onset is abrupt, the [illegible] tioned symptoms develop in rapid succession. There is often an absence of distinct exophthalmus and of a thrill or *bruit* over the thyroid, the typical crisis of exophthalmic goiter is wanting. The basal metabolic rate is always increased. It is apt to occur past middle age.

(*f*) ***Exophthalmic Goiter*** (Graves' disease, Basedow's Disease, Thyroid Toxicosis) *Definition:* Exophthalmic goiter is a constitutional thyroid toxemia, characterized clinically by instability of the nervous system, diffuse enlargement of the thyroid gland, exophthalmus, tremor, tachycardia, hyperhidrosis, gastrointestinal disturbance, dermographia and increased basal metabolic rate. It is characterized pathologically by parenchymatous hyperplasia of the thyroid, hyperplasia of the lymphatic system and thymus, hypocholesteremia and increased iodine content of the blood.

Etiology: There is a hypersecretion of thyroid hormone which probably contains a toxic substance. The following may be factors in upsetting the thyroid balance: (*a*) Heredity, which may either transmit the disease or transmit a predisposition to it which, in the presence of exciting factors such as worry, fright, local or systemic infections, or mental and physical strain, will bring forth the disease in an active stage; (*b*) disease of other endocrine glands and particularly when the thyrotropic hormone of the pituitary is affected; (*c*) psychic trauma, physical strain and overwork, even in the absence of any hereditary predisposition, and (*d*) it may occur in the absence of any definite or discoverable cause, possibly due to hypersensitivity of the various tissues of the body to the thyroid hormone, or a deficiency of thyroid antihormone. Women are more prone to it than are men. Exophthalmic goiter is most prevalent during the second and third decades. It is often characterized by periods of remissions and recrudescence.

Symptoms and Signs: These depend upon the severity of the disease and whether the patient is in a crisis or in a state of remission. Mild cases naturally show fewer and milder signs. During

Differential Table Between Toxic Adenoma and Exophthalmic Goiter

Toxic Adenoma *(Hyperthyroidism, Secondary Toxic Goiter, "Basedowified" Goiter)*	Exophthalmic Goiter *(Graves' Disease, Basedow's Disease, Parry's Disease, Flajani's Disease, Hyperplastic Goiter, Dysthyroidism Thyrotoxicosis)*
1. Patient is usually of middle age.	1. Patient is usually a young adult
2 Goiter present years before onset of symptoms.	2. Goiter often absent; if present, is of recent occurrence.
3 Goiter is essentially adenomatous, often nodular in shape, and usually large, nonpulsating, noncompressible, without thrill or bruit.	3. Goiter is essentially hyperplastic in nature, rarely large, usually a symmetrical fullness, often pulsating, compressible, and presents thrill and bruit.
4 Exophthalmus and expression of chronic fright rare, eye signs not prominent	4 Exophthalmus and expression of chronic fright with characteristic eye signs are usually present
5. Tachycardia not extreme, often materially slowed by sleep or digitalis.	5. Tachycardia more pronounced, not appreciably slowed by sleep or digitalis
6 Hypertension and myocardial degeneration common.	6. *Hypertension not common, myocardial* degeneration occurs late in the disease.
7. Tremor often absent, if present, is coarse and atypical.	7. Tremor nearly always present and typical.
8. Mental symptoms relatively mild	8 Mental symptoms relatively prominent, with occasional major psychoses
9 No tendency to gastrointestinal crises.	9. Tendency to gastrointestinal crises
10. Dermographia often absent, when present, is not intense	10. Dermographia constant and usually intense; other skin lesions common
11 Loss in weight comparatively slow.	11. Loss in weight comparatively rapid
12. Symptoms may be produced in a normal person by administration of thyroid extract or thyroxin	12. Syndrome not produced by administration of thyroid extract or thyroxin unless predisposition exists
13 *Surgical interference with the thyroid* eminently successful; usually no recurrences *or regeneration, as mass is* encapsulated.	13. *Surgical interference with the thyroid* not always successful, recurrence be*cause of regeneration may occur* as the mass is unencapsulated
14 Remissions do not occur.	14 *Remissions and exacerbations common*

a crisis all signs are greatly intensified; and fever, diarrhea, hyperhidrosis, tachycardia or auricular fibrillation, and other toxic 'manifestations are greatly aggravacated. ***A typical case of average severity will present the following:***

(1) The *general appearance* is that of fright or great anxiety; the patient is restless, impatient and cannot find a place for *himself. The face is flushed* or covered with perspiration.

(2) The *eyes* are staring or protrude (exophthalmus). *This may be unilateral* but is most often bilateral. Very rarely typical exophthalmus may be absent. A number of eye signs usually accompany the exophthalmus, of which the most common are: (*a*) *Von Graefe's Sign:* Failure of the upper lid to follow the downward movement of the eyeball; (*b*) *Moebius' Sign:* Failure of convergence of the eyeballs when looking downwards;

(*c*) *Stellwag's Sign:* Inhibition or lessening of the wrinkling reflex; (*d*) *Joffroy's Sign:* Absence of wrinkling of the forehead when the eyes are rolled upward as far as possible; (*e*) *Dalrymple's Sign:* Widening of the palpebral fissures, (*f*) *Riesman's Sign* Audible bruit heard over the eyeball; (*g*) *Loewy's Sign:*

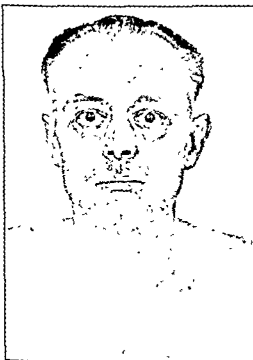


Fig 13—Exophthalmic goiter The B M R was plus 90 The thyroid was palpable and pulsating All other classical signs were present Patient died within 24 hours after thyroidectomy in crisis

Prompt and lasting mydriasis when two drops of 1 1000 epinephrine solution is instilled in either eye

(3) *The Neck:* There is usually present a symmetrical fullness; often it is a large yielding pulsating mass; occasionally no definite thyroid enlargement is visible The thyroid may be easily palpated by grasping the lower part of the neck between the thumb on one side and index and middle fingers on the other side of the anterior bellies of the sternocleidomastoid muscles when the chin is raised, particularly during the act of swallowing. The gland may be impalpable in substernal thyroid. Occasionally the thyroid gland may not be enlarged, though there may be hyperactive thyroid tissue in aberrant positions. In addition to the large thyroid, there are visible pulsations of the vessels of the neck, and a generalized erythema of the skin of the neck and of the adjacent upper portion of the chest. A thrill may be felt or a *bruit* heard over the thyroid

(4) *Cardiovascular System·* Tachycardia, during excitement and also when at rest, associated with dyspnea is an early sign; in the more advanced cases there occur signs of cardiac decompensation and various arrhythmias, particularly auricular fibrillation.

(5) *Gastrointestinal Symptoms.* The appetite is usually good, but notwithstanding that, there is a persistent loss of weight. Nausea, vomiting and diarrhea are usually present at a crisis.

(6) *Cutaneous Manifestations:* Flushing of the face and neck and moisture of the skin with profuse sweating on emotion or mild exertion are nearly always present The skin is generally soft, pliable and smooth, often there are brownish pigmented areas, papules, pustules and itching are frequently present. The patient usually feels warm

(7) *Tremors·* There is a decided fine tremor noticed in the outstretched hand and a general muscle tremor is perceivable over the entire body.

(8) *The Genital System·* Menstrual disturbances such as dysmenorrhea, oligomenorrhea, amenorrhea, metro- and menorrhagia may occasionally occur.

Libido is poor and sterility is common. In men there often occurs lack of libido or potentiality.

(9) *Basal Metabolic Rate:* This is most often increased from plus 30 to 90 or over. In rare cases, the basal metabolic rate is not markedly elevated. A rough guess of the B M.R. may be had by employing the *Read formula*: The pulse rate is added to the pulse pressure, and from the sum, 111 is subtracted. Thus, for pulse rate 90, pulse pressure 60—90 plus 60, minus 111, equals plus 39.

(10) *Blood:* Secondary anemia and a tendency to lymphocytosis are usually present.

(11) *Blood Pressure:* The systolic pressure is usually elevated and the diastolic is lowered so that there is an increased pulse pressure. The systolic pressure rises sharply and all toxic symptoms become intensified by the administration of epinephrine.

(12) The *"Goetsch Test"* is positive *In well-marked cases this test should* not be used This test is carried out as follows: Five to seven and a half minims of 1.1000 epinephrine solution is given hypodermically. Every five minutes during the next hour it will be noted that the systolic pressure has risen from 10 to 50 points, the pulse rate is increased from 10 to 20 beats per minute. There is also an increase of nervousness, tremors, sweating and flushing, though at times there may be pallor of the face. The pupils remain dilated for from one-half to one hour.

Other laboratory examinations will usually show a decrease in the blood cholesterol, an increase in the blood iodine content, at times to as high as 30 gamma per cent, and a slight hyperglycemia and low blood calcium

(13) *The Urine:* Increased frequency by day and night, frequent glycosuria, moderate albuminuria, and increased excretion of iodine, and of nitrogenous products are present in the majority of cases.

(14) *Drug Tolerance:* There is an increased tolerance to quinine (Bram), physostigmine and ergot, and a decreased tolerance to epinephrine and other sympathomimetic drugs.

Atypical Forms of Exophthalmic Goiter: While the symptoms just enumerated are found in typical cases of exophthalmic goiter of moderate severity, there are cases in which some of the cardinal signs are wanting Occasionally there may be an absence of exophthalmus; in some cases the thyroid may not be palpably enlarged, and in other cases the B.M.R. may not be elevated above the usual normal values In children, in the senile and in the obese, many of the signs may be absent though the majority are present

Masked Hyperthyroidism: This condition is so called because there may be an absence of exophthalmus, and of visible nervousness. This condition is usually found in elderly people. They are apathetic; are easily fatigued; have a slight staring of the eyes; have a sense of warmth; an increased basal metabolic rate, and frequently they have diarrhea. Tachycardia may be present or absent, but the heart rate is easily accelerated by moderate exertion.

Hypothyroidism or Thyroid Insufficiency (Myxedema, Cachexia Strumipriva, Gull's and Ord's Disease, Childhood Myxedema, or Cretinism)

Hypothyroidism is a condition brought about by thyroid insufficiency, that is.

the lack of thyroid secretion. This is characterized in the young by the retardation of physical and mental development and the diminution of metabolic activity; and in the adult by slowing of all metabolic activities and by mental and physical retardation The amount of retardation depends upon the age at which the thyroid becomes hypoactive or inactive, and on the degree of its hypoactivity or inactivity When thyroid inactivity occurs at birth or soon thereafter, it results in cretinism, when the thyroid becomes inactive or hypoactive in older children or in adults, then the condition is variously known as myxedema, cachexia strumipriva, Gull's disease, or Ord's disease Milder types of hypothyroidism bear no specific name Hypothyroidism may be primary or secondary. *Primary hypothyroidism* may be caused by a diseased thyroid or by insufficient thyroid tissue which causes either a deficiency or lack of thyroid hormone It is also quite possible that an "insufficient" amount of thyroid hormone may be due to deficient thyroid stimulation by the anterior pituitary thyrotropic hormone

Secondary hypothyroidism may be due to disease of the gonads, wasting diseases, starvation or other diseases that either limit the secretion of thyroid hormone or interfere with the absorption of the thyroid hormone by the tissues Another probability is that there may be an overproduction of thyroid antihormone.

Adult Myxedema, *Symptomatology and Diagnosis:* Hypothyroidism, myxedema and cachexia strumipriva are adult types of diminished or absent thyroid activity The commonest phenomena in a well-marked case are as follows (1) Pallor, (2) subcutaneous swelling; (3) rough, lusterless, dry and cool skin imparting to the touch the sensation of dead rubber, (4) coarse, dry and scanty growth of hair, (5) general listlessness, (6) supraclavicular fat pads; (7) associated nephritis, (8) bradycardia; (9) subnormal temperature; (10) dull, listless and stupid facial expression, the features being almost



Fig. 14—Myxedema

immobile, (11) puffy lower eyelids, (12) thickened lips, tongue and nose; (13) dull, coarse and monotonous voice; (14) slow body movements; (15) staggering gait; (16) nervous symptoms, such as headache, slow perceptive powers, alterations of temper and perverted taste and smell; (17) aches and pains in the extremities, (18) the blood shows a definite anemia and, because of the associated skin pallor, may resemble pernicious anemia; (19) the blood cholesterol is high (300 to 700 mg.), (20)

the blood iodine is low; (21) the basal metabolic rate is abnormally low and may vary from minus 20 to minus 40, and (22) there may be a hypochlorhydria or an achylia gastrica

Mild cases of hypothyroidism are often found near or past the menopausal age in both women and men. It is manifested by fatigability, various aches and pains, digestive disturbances, thinning of the eyebrows, secondary anemia, a decreased basal metabolic rate, a low gastric acidity, and an increased blood cholesterol

Cretinism: Cretinism may be defined as a state of continuous and abnormal infancy due to arrested physical and mental development which began before or soon after birth as a result of congenital thyroid insufficiency Immediately after birth there are, as a rule, few or no signs of athyroidism. The newborn infant, in most instances, appears normal, possibly because *in utero,* the fetus, being nourished by the mother's blood, does not suffer from his own thyroid insufficiency. Also, as long as he is breast fed by a mother whose thyroid gland is normal, the infant will show no signs of thyroid deficiency. After weaning, or in an artificially fed child, the lack of thyroid secretion manifests itself as soon as the child reaches a stage where he has to depend upon his own hormones for physical and mental development

There are two types of cretinism, sporadic and endemic.

Sporadic cretinism: This may occur in an individual not descended from cretins as isolated cases in localities where cretinism does not prevail.

Endemic Cretinism: This is often familial and is indigenous to certain locations, as in the so-called goiter belts of this country and abroad. The endemic cretin differs from the sporadic in that the endemic cretin is generally not quite as helpless as the sporadic, his growth is not as stunted, his mentality is not quite as blank, and his genitals are not as hypoplastic as are those of the sporadic cretin. The endemic cretin often has a large colloid goiter or a useless thyroid such as may be found in his mother or father. The sporadic cretin is usually in a state of continuous infancy, is helpless, stupid and ungainly



Fig 15—Cretin Age 34 years Complete athyroid cretin.

Characteristics of Severe Cretinism: The head is large and rounded,

the facies are coarse and puffy; the complexion is sallow or pasty; the eyelids are puffy; the nose is thick and its bridge is depressed; the lips are thick and dry, and saliva often drools from the mouth; the tongue is thick, large and broad. The teeth are poorly developed; the neck is short, the trunk is rounded and longer than the extremities, there are fat pads over the shoulders The abdomen is large and protruding, often showing an umbilical hernia. The extremities are poorly developed, usually cold and cyanosed; the long bones show retarded development The hands are round and puffy and the fingers are broad and square at the tips The hair is coarse and straggly. The mentality is greatly retarded, deaf mutism is common; and the reaction to stimuli is exceedingly slow. Most frequently there is imbecility.

Cretinism appearing during early childhood is practically childhood myxedema, showing, in addition to the general signs of myxedema, retardation of the ossification centers, particularly in the carpal bones Sporadic cretinism and myxedema respond to thyroid medication, while response to the same treatment in endemic cretinism is poor Endemic cretinism may be prevented or its severity ameliorated by the early administration of iodine.

The Pineal Gland

Anatomy and Physiology of the Pineal Gland

The pineal gland is a small cone-shaped body in contact with the third ventricle of the brain It is composed of characteristic pineal cells, neuroglia and connective tissue It is richly supplied with blood vessels and nerves, and often harbors brain sand, and occasionally, small cysts From the appearance of granules in the protoplasm of its cells and because of its rich blood supply, it is assumed that the pineal body is an active endocrine gland No pineal hormone has as yet been isolated Experimental studies have so far proven that the gland is not a vital organ

The Function of the Pineal Body: Extirpation and feeding experiment upon animals are inconclusive Clinical observation suggests that the pineal body is intimately connected with sexual maturity One group of observers believes that the pineal body acts as a checkrein to the gonads, retarding their development until bodily maturity has taken place. Another group believes that the pineal body stimulates sex maturation There is no definite knowledge at present with regard to pineal function, though the consensus is in favor of the theory that the pineal body acts as a checkrein to the gonads, inhibiting their premature development

Diseases of Pineal Origin

Tumors of the pineal body may cause, in addition to neighborhood pressure symptoms, precocious puberty

Macrogenitosomia Precox

This syndrome in boys is often associated with pineal tumor It is characterized by rapid growth of the skeleton up to the sixth year, then growth slows or stops because of premature epiphyseal union. During the period of rapid growth there is also precocious genital development (premature adulthood); the genitalia become large; hair develops upon

the mons, under the arms and on the face. The *mentality* matures; the voice becomes low pitched, and physical development with obesity becomes marked. In the presence of a pineal tumor, intracranial pressure symptoms such as headache, blindness, paralysis and hydrocephalus develop sooner or later.



Fig 16—Pineal tumor with hydrocephalus, age 5 years. Note general development, large head and pubic hair (Philadelphia General Hospital)

The Thymus Gland

Anatomy and Physiology of the Thymus Gland

The thymus gland is composed of lymphoid tissue. It contains two lobes, each of which is made up of lobules bound together by connective tissue The cortex consists of closely packed lymphocytes and the medulla contains a reticulum of large branched cells, few lymphocytes and the concentric corpuscles of Hassall. The thymus gland is situated in the mediastinum, is bordered on either side by the lungs and is in close relation to the pneumogastric, phrenic and recurrent laryngeal nerves and the large blood vessels. The gland is largest during infancy and early childhood, attains its full size at or about the second year and,

according to Hammar, becomes involuted at puberty (11th to 15th year)

Hormone: No hormone has as yet been isolated from the thymus However, an extract made of thymus tissue produces excessive growth in immature animals.

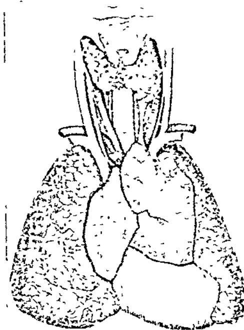


Fig. 17—Thymus gland of full-time fetus (Engelbach's "Endocrine Medicine," Charles C. Thomas, Springfield, Ill)

Pathology: The thymus gland may be the seat of tumor or cyst, or it may fail to involute at the proper time (persistent thymus). Enlargement of the thymus may occur because of disease elsewhere such as exophthalmic goiter, Cushing's syndrome, hypogonadism, castration, Hodgkin's disease, leukemia, septicopyemia or myasthenia gravis The thymus gland may also be affected by syphilis, tuberculosis, or it may become infested by parasites. Atrophy of the thymus is seen in marasmus, in profuse hemorrhage and in inanition

Physiology: The thymus, like the pineal, is a gland of childhood; both involute at or about puberty and neither gland has so far yielded a specific hormone. It is believed that the thymus is concerned with the growth and development of the body, the gonads and the osseous structures. The administration of thymus extracts to immature rats either directly, as by Asher, or through successive generations, as by Rowntree and his co-workers, enhanced their growth; while ablation of the gland retarded their growth. The exact rôle the thymus plays in the physiology of the organism is not known; its retrogression at the age of puberty, when the sex glands are fully developed and its persistence in hypogonadism are significant of a gonad-thymus relationship probably mediated through the anterior pituitary, the thyroid and the suprarenals

Diseases of Thymus Origin

Though the functions of the thymus gland are not definitely known, there are a number of constitutional anomalies characterized by definite stigmata that occur sufficiently often to indicate that they may be of thymus origin, or that the thymus plays an important rôle in their production

Hyperthymism

Status Thymicolymphaticus (status hypoplasticus, lymphatism) : Status thymicolymphaticus is a constitutional anomaly characterized by definite stigmata It is generally congenital, but may be acquired during childhood. The clinical picture of this condition varies with the age of the individual and the degree of involvement.

In children the following is characteristic: *The child is delicately molded,* is slender and graceful. The skin in some is soft, delicate, of velvety texture, and *faintly cream colored; in others,* it may be dead white, lusterless or pasty in appearance; or it may be unusually shiny. *The surface of the body remains* covered with lanugo beyond the usual age. The hair upon the head is soft and often curly. The face presents the "angelic appearance"; the eyes are round, light blue or brown; the lashes are long and curl upward. The nose is small; the mouth usually pouts; the cheeks are rounded and flush or pale readily There is general lymphatic hyperplasia in the neck, axillae and groin. The tonsils and adenoids are enlarged and the spleen is often palpable. The genitals are hypoplastic. The child is generally timid, irritable, has a high pitched voice and has a greater susceptibility to upper respiratory *infections and various skin rashes;* is sensitive to protein inoculations, and is allergic to a vast variety of substances.

Among adolescents and adults, three types of lymphatism may be considered (*a*) The obese or *hypothyroid type;* (*b*) the *thin* or *hyperthyroid type, and* (*c*) the well-nourished or classical type This division is purely arbitrary and is based upon the corpulence of the individual and his general behavior

(*a*) ***The obese or hypothyroid type*** is soft, flabby and bloated, has coarse features, and is mentally and physically sluggish.

(*b*) ***The thin or hyperthyroid type*** is very thin, often emaciated and has small features, a *long neck,* a *cylindrical* body and long lower extremities. The genitalia are well formed but their function is below normal. This type is usually alert and restless.

(*c*) ***The well-nourished or classical type*** is usually somewhat below normal in height and has a youthful appearance. The palate is high arched

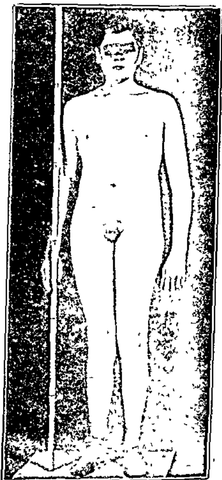


Fig. 18—The well-nourished or classical type of hyperthymism, with extreme hypogonadism, showing infantile-shaped body, long slender extremities and rudimentary genitals (Phila. Gen Hosp.)

(torus palatinus). *The teeth are* bluish white in color and irregular. The central incisors may be large and lateral *incisors may be rudimentary* while the canines are usually small and may resemble the incisors. The neck is short;

isolated lymph glands of the anterior and posterior chains are palpable as are also some in the supraclavicular fossa.

The thorax is slender and rounded, resembling in shape and conformity that of the child. The upper and lower extremities are rounded and well shaped, the fingers are long, appear sensitive and are extremely flexible so that they can easily be bent backwards, suggesting a double jointedness Most of the joints of the body are lax and may be easily dislocated or contracted

The hair upon the mons is triangular in shape, resembling the female type of distribution, *i. e.*, the base line upward and the apex pointing downward The hair in the axillae and on the extremities is sparse or entirely absent. The genitals are often poorly developed, hypospadia and unilateral or bilateral cryptorchism are not uncommon.

The female of this type is also characterized by the appearance of plumpness, softness of the skin, irregular dentition, enlarged lymph glands, loose jointedness and sparse distribution of hair The genitalia are hypoplastic; the clitoris is often enlarged; menstrual disturbances, such as amenorrhea, hypomenorrhea and dysmenorrhea, are common; and occasionally there may be excessive bleeding at irregular intervals.

This type is usually associated with a marked degree of genital disturbance and with anterior pituitary and adrenal medullary hypofunction

Characteristics Common to All Types of Status Thymicolymphaticus: While the three types mentioned, namely, the obese, the thin and the well nourished, exhibit certain individual characteristics, yet there are a number of clinical manifestations common to all of them which justifies their grouping into a general classification. The common characteristics of all types of status thymicolymphaticus are: (1) An enlarged thymus gland which is not always demonstrable during life; (2) hyperplasia of the lymphatic structures; (3) a youthful appearance; (4) sparse hair distribution; (5) hypogenitalism; (6) hypoplasia of the cardiovascular system; (7) anomalies of the gastrointestinal tract; (8) vascular hypotension; (9) low basal metabolic rate; (10) easy fatigability; (11) a relative lymphocytosis; (12) low carbon dioxide tension; (13) a tendency to asthma, hay fever and other protein sensitivity, (14) a tendency to sudden unexplainable death or death due to adrenal or intracranial hemorrhage or to coronary disease; (15) greater susceptibility to infection and greater death rate from acute infection; (16) evidence of vagus disturbances, and (17) psychic disturbances. Their mentality may be normal but their behavior is often much like a spoiled "only child" They are selfish, obstinate and negativistic. Some may possess ungovernable tempers and may be unreasonable. Another of their characteristics is an inability to apply themselves to certain situations, to sustained effort or to creative work Their accomplishments are usually the result of imitation rather than of original effort. Notwithstanding the innate handicap of these unfortunates whose disability is not of their own choice or making, many of them, with proper training and wise supervision, may be developed into normal individuals and useful members of society

Other Possible Hyperthymic Conditions

Other conditions attributed to hyperthymism are:

(1) *Mors Thymica:* This is where death occurs in a child suddenly and *without any* apparent provocation. The existence of such a specific type is questioned.

(2) *Thymic Stridor:* This is difficult or stridulous breathing occurring at certain times, particularly after excitement or crying (rare).

(3) **Thymic Asthma:** The occurrence of bronchial asthma is at times attributed to an enlarged thymus, but it is doubtful whether the thymus enlargement is responsible for these conditions.

(4) **Myasthenia Gravis:** This is thought to be due to thymus involvement. It is characterized by nasal speech, ptosis, exhaustion and fatigability of the striated muscles (SEE: p. 881).

Hypothymism

Timme described a syndrome due to premature involution of the thymus. The individuals are stockily built, have a compact frame and short stature. Epiphyseal ossification and maturation occur prematurely. The secondary sex characteristics may appear during childhood The permanent teeth appear early but are irregular and the blood pressure is generally high The mentality is a combination of childhood stubbornness and adult resourcefulness; they are mean, cruel and easily angered.

The Parathyroid Glands

Anatomy and Physiology of the Parathyroid Glands

The parathyroids are four in number, situated behind and intimately connected with the thyroid gland. Accessory parathyroids are fairly common and may be found in positions close to the regular parathyroids, as in the thyroid gland, the thymus and in other structures of the neck or upper chest. They measure approximately 6 x 3 x 2 mm. The parathyroids are made up of two types of cells (*a*) The chief cells, which are polygonal in shape and are most numerous, and (*b*) the oxyphil cells, which are larger and contain deeply staining nuclei.

Hormone: The parathyroids elaborate a hormone which influences the metabolism of calcium and phosphorus. Parathyroid activity is believed to be under the influence of the parathyrotropic hormone of the anterior pituitary lobe Calcium metabolism is also influenced by vitamin D and by the actinic sun rays which act synergistically with the parathyroids. The parathryoid hormone (parathormone) was isolated and made available for clinical use by J. B Collip

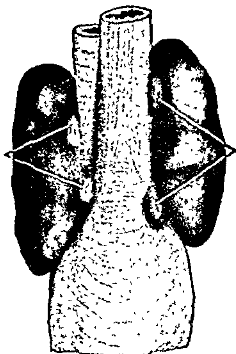


Fig. 19—Human thyroid and parathyroid. Posterior view. (From Zuckerkandl)

A unit of parathormone is considered to be 1/100 of the amount required to raise the blood calcium about 5 mg. to the 100 cc. of blood in a dog, weighing 20 kilograms, within 15 hours after injection The international dog unit is one-fifth the strength of the Collip dog unit. The normal blood serum calcium is between 9 and 11 mg. to 100 cc. of blood; the normal serum phosphorus between 3 5 and 4 mg. to 100 cc of blood

Diseases of Parathyroid Origin

Disease of the parathyroids may cause either hyper- or hypoparathyroidism In hyperparathyroidism there occur conditions that are associated with hypercalcemia, and in hypoparathyroidism conditions occur in which hypocalcemia is the dominant factor

Hyperparathyroidism

Hyperparathyroidism is recognized by an increase of serum calcium, often ranging from 12 to 20 mg to 100 cc of blood and a decrease of serum organic phosphorus to 1 5 to 3 mg. to 100 cc of blood. The nervous system becomes much depressed; the heart slows; and there occurs hypotonicity of the muscular system with pain in the limbs, so that walking or muscular exertion is difficult. The kidneys become affected; the urine contains large amounts of calcium and phosphorus; renal stones are common. Gastrointestinal symptoms, such as anorexia, nausea, vomiting and constipation are prominent. The osseous system shows characteristic changes All the bones of the body are decalcified (osteoporosis) and many undergo fibrosis, bony tumors and cysts may occur in the long bones or in any of the other bones of the body. Spontaneous fractures may occur in the long bones, in the pelvic bones and at times in other bones, causing deformities and shortening of the stature. The bones of the jaw and spinal column may likewise become affected Because of the fibrous cystic degeneration of the bones, this condition is known as *"osteitis fibrosa cystica"* or "Von Recklinghausen's disease of the bones." Minor degrees of hyperparathyroidism exist in which the symptoms are less pronounced. Renal calculi or otosclerosis may occur in hyperparathyroidism without showing severe bone decalcification.

Hypoparathyroidism

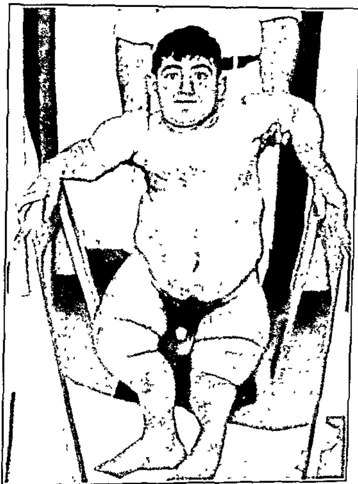
Hypoparathyroidism causes a diminution of calcium in the blood and since calcium is a nerve sedative, a deficiency of blood calcium will cause neuromuscular hypersensitivity. This is recognized by hyperexcitability of the entire nervous system which produces sensory, motor and autonomic nervous system phenomena causing, among other manifestations, tonic spasms of the skeletal muscles with generalized convulsions The spasms of the extremities are usually bilateral and may simultaneously affect all four extremities or, as occasionally happens, only a single extremity or isolated group of muscles may become affected. Gastrointestinal symptoms, such as anorexia, vomiting and diarrhea, are common Nervous irritability, insomnia, perversion of temper and other signs of instability of the nervous system are early manifestations. Other signs of hypocalcemia are defects in the enamel of the teeth, brittle and grooved nails, juvenile cataracts, alopecia, retarded growth, hypotension and a tendency to asthma.

Hypoparathyroidism may occur because of injury or disease of the parathyroids or because of their extirpation

during a thyroidectomy or other operation in that region. The most familiar syndrome resulting from parathyroid insufficiency is tetany.

Tetany

This is described as a symptom complex characterized by neuromuscular hyperexcitability, which is manifested by excitement, irritability, poor muscle control, twitchings, tonic spasms of the muscles of the extremities that produce characteristic deformities, such as the obstetric hand and other attitudes and general convulsions. Tetany is a symptom in hypoparathyroidism *when the blood* shows a decided diminution in its calcium content. In these conditions, the serum calcium may fall to as low a level as 7, 6, or less mg. to the 100 cc. of blood. Tetany may also occur in hypocalcemia not of parathyroid origin, because a low blood calcium is found in conditions other than frank parathyroid disease, *i. e., the lack of vitamin* D and of actinic sun rays. Finally, tetany may be produced by a number of conditions in which both the parathyroids and blood



calcium are normal. Such is seen in alkalosis which may result from the ingestion of excessive quantities of alkalies, from the lack of HCl in the stomach caused by vomiting, or by other conditions, also, from the loss of CO_2 by crying, or

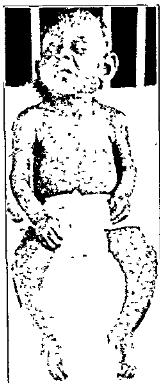


Fig 21—Tetany—Hypoparathyroidism Note the carpopedal spasm and rotation of head.

by other conditions causing hyperventilation of the lungs

Etiology: The etiology of tetany is varied; it may be due to parathyroprива, to hypocalcemia, to alkalosis, to pregnancy, lactation and menstruation, to vitamin D and sunshine deficiency, to hyperventilation of the lungs and to gastrointestinal diseases It is also found in the newborn, in infants, in rickets, in infections and in certain poisonings Tetany may also be idiopathic and may occur in certain localities, in certain occupations and also in epidemics. It is more prevalent during the cold winter months, possibly because of the lack of actinic sun rays and of dietary essentials

Irrespective of its etiology, the clinical manifestations of tetany are identical, and are an aid to its diagnosis; certain signs are described which will help to identify the condition by demonstrating the hyperexcitability of the nervous system.

Tetany may occur as active or latent Active tetany presents all or many of the signs of neuromuscular hyperactivity Latent tetany has the neuromuscular excitability under control or it is masked, and may be brought forth by some provocation, such as anger, excitement, sickness or by mechanical, electrical or chemical irritation.

Diagnosis: The special diagnostic signs of tetany are as follows

1 *Erb's Phenomenon* Increased reaction of the motor nerves to the galvanic current (constantly present)

2 *Hoffman's Phenomenon* Increased excitabiltiy of the sensory nerves to electrical stimulation.

3 *Trousseau's Phenomenon* The ulnar nerve is usually used Contracture of the fingers is produced (obstetrical hand) in latent tetany, by the application of a tight ligature around the upper arms (Of great diagnostic value)

4 *Chvostek's Sign* This consists of three related groups of contractures depending upon the degree of tetany. In severe tetany, light percussion in the region of the external auditory meatus (pes anserinus) causes contractions of the muscles of the whole side of the face closing the eyelid, contracting the ala nasi and the corner of the mouth on the

side percussed. These signs may at times be brought out by stroking of the skin near the auditory meatus. In moderate tetany, tapping over the zygoma produces contraction of the ala nasi and the muscles of the corner of the mouth. In mild tetany, percussion over the zygoma or masseter muscle will cause only slight twitching of the angles of the mouth.

5. *Schultze's Tongue Dimpling Sign* A dimple is formed upon the protruded tongue when it is sharply struck with a pointed instrument or finger tip

6. *Schlesinger's Leg Phenomenon* Flexing the hip joint when the leg is extended at the knee causes painful spasms of the leg

7. *The Arm Phenomenon of Pool:* Sudden forcible abduction of the arm causes contractures of the muscles of the arm.

8. *Kashida's Phenomenon:* Hyperexcitability of a nerve is induced by the application of a hot or cold irritant.

9 *Injection of a Foreign Protein:* This may initiate an attack of tetany.

10 *Hypersensitivity to Adrenalin or Pilocarpine:* Adrenalin when injected will cause a sharp rise of blood pressure, tachycardia and blanching due to constriction of the superficial vessels. The injection of pilocarpine will cause excessive sweating, "goose skin," increased salivation, lacrimation, flashes of heat and a congested feeling in the head.

The Adrenal Glands

(The Suprarenal Glands)

The adrenal glands, two in number, are situated each above its respective kidney retroperitoneally. They are extremely vascular and are well supplied with lymphatics The two component parts, *i. e*, the cortex and the medulla arise from different layers of the blastoderm. The cortex springs from the mesoderm and the medulla is of neuroblastic origin (ectoderm).

The Adrenal Cortex

Functions of the Adrenal Cortex: (1) The cortex is essential to life; destruction of all cortical tissue causes death speedily, while the administration of cortical extract in a decorticated subject maintains life over a long period.

(2) It maintains vital influence upon body function and metabolism.

(3) It maintains a normal level of sodium and prevents the accumulation of high level of potassium in the blood

(4) It assists the liver in storing glycogen and in converting protein into dextrose.

(5) It assists in maintaining muscle tone and endurance.

(6) It influences gonad development and function. The activities of the adrenal cortex are carried out by its hormone, the production of which is stimulated by the adrenalotropic hormone of the anterior pituitary body.

Hormone, etc.: The cortical hormone is variously known as interrenalin, cortin, adrenocortical hormone, eschatin (commercial name) and interrenin It is the vital hormone of the cortex It therefore maintains life and vigor or when it is administered to individuals suffering from adrenal cortex hypofunction it restores vigor and normal metabolic processes. The administration of the cortical hormone to healthy individuals does not produce symptoms of hypercortical activity.

Substances other than the cortical hormone found in the adrenal cortex are. Cartilactin, suspected of being a galactogogue; ascorbic acid, known also as cevitamic acid or hexuronic acid, which *is identical with vitamin C, and cardais*sin, a circulatory stimulant of indefinite origin. The adrenal cortical hormone is also produced synthetically in the laboratory as Desoxycorticosterone Acetate and dispensed commercially under various trade names.

Diseases of the Adrenal Cortex

Diseases of the adrenal cortex may cause hyperactivity, as seen in cases of certain cortex tumors, hyperemia, and hypertrophy; and hypoactivity, as seen in cases of partial destruction of the cortex by syphilis, tuberculosis, or other destructive lesions

Cortical Hyperfunction: The clinical syndrome produced by hyperfunction of the adrenal cortex depends upon the period of life in which the cortex becomes affected

Pseudohermaphroditism is often due to congenital tumors of the adrenal cortex.

Pubertas precox is found in childhood affections (tumors) of the suprarenal cortex. The disease is characterized by premature bodily and sexual development. The individual may be *either stout or thin, is quite hairy and* matures early.

Virilism occurs in the adolescent or adult females The individual loses her feminine appearance and assumes masculine mannerisms and characteristics *She may become excessively stout or* thin. Hair appears upon the face and body, the pubic hairs assume the masculine distribution; the voice becomes low pitched, the gait develops a masculine stride and swing; the shoulders appear broad, and the pelvis seems narrow. Menstruation either ceases or menorrhagia, metrorrhagia, or oligomenorrhea develops

Cortical Hyperactivity in the adult male is characterized by hyperemia, some adiposity, hypertension and gonad hypofunction

Cortical Hypofunction: This will produce weakness, pigmentation, hypotension, gastrointestinal disturbance, hy-



Fig. 22—Right and left adrenals
(Engelbach's "Endocrine Medicine," Charles C Thomas, Springfield, Ill)

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tal or acquired Congenital tumors may be benign adenoma (rare), which may undergo malignant change, and primary malignant tumors such as sarcoma. Congenital cortical tumors may be responsible for pseudohermaphrodism. Acquired tumors may originate during childhood, adolescence or adulthood; they may be benign, primary malignant or metastatic. Childhood tumors may cause pubertas precox. Adolescent or adulthood tumors may cause virilism, heterosexual changes, hypertrichosis or other signs of hyperadrenia. If the tumor destroys the adrenal cortex, signs of hypoadrenia may become manifest

Hypernephroma (Grawitz tumor). This is a tumor of the kidney which is believed by some to develop from adrenal cortical "rests"; others doubt this origin It is a malignant neoplasm which may invade the adrenal and give rise to symptoms of hyperadrenalism, *i e*, hypertrichosis, macrosomia and virilism. It usually metastasizes to the lungs liver and other organs and causes weakness, emaciation, pulmonary and gastric symptoms and occasionally hematuria

The Adrenal Medulla

The adrenal medulla appears to be the less important of the two suprarenal structures. The results obtained from animal experimentation show that the medulla is not essential to life Animals, whose entire medullary tissues of both adrenals were ablated, continued to live for a long period and showed no ill effects Whether this is due to compensatory work done by the other chromophil tissues in the absence of the adrenal medulla, or whether the medulla is unessential is not definitely known.

Function of the Adrenal Medulla: The actual function of the adrenal medulla is not definitely understood. It is believed by some that the medulla is a reserve organ, functioning only when called upon by unusual emotional circumstances such as fright, anger, and impending physical injury or death. Under such circumstances, the medullary hormone is secreted in sufficient quantities to stimulate the circulation, tone up the nervous system, and mobilize sufficient muscle glycogen so that the individual is ready for offensive or defensive action

Hormone: The hormone elaborated by the adrenal medulla is known as epinephrine, adrenalin, suprarenal, adrenine, adrin, and probably by several other names. It has a definite formula and is produced synthetically Its physiologic action is stimulation of the sympathetic division of the autonomic nervous system. It will therefore cause increased heart rate, elevation of blood pressure, constriction of the superficial blood vessels, dilatation of the pupils and, often, hyperhidrosis The medullary hormone has a beneficial effect upon bronchial asthma, upon allergic states, such as urticaria, hay fever and upon certain protein reaction phenomena. It is also employed as a local anesthetic or is used in conjunction with a local anesthetic so as to enhance the action and control bleeding during the operation. It has a tendency to mobilize sugar in the blood and may therefore be used in states of acute hypoglycemia For systemic effect adrenalin is active only when given hypodermically, intravenously, intraperitoneally and intracardiac. It also has a mild systemic effect when applied to mucous and serous surfaces, *i. e*, the nose, the conjunctivae, under the tongue, in the rectum, and in the vagina

poglycemia and disturbed renal function leading to severe *intoxication.*

Addison's Disease: *This is the specific entity caused by partial destruction* of the adrenal cortex. The severity of the symptoms and the course of the disease depend upon the amount of cortical tissue involved and the *rapidity* with which it is destroyed. The disease is characterized by adynamia or severe asthenia on least exertion and pigmentation of the skin and the mucous membrane. The entire skin may become darker; dark areas are noted particularly on the hard palate, on the side of the tongue, and on parts of the body exposed to the sun and to irritation, such as the face, hands, scapulae, waistline and around the arms. The blood pressure is very low, the systolic pressure may be between 60 and 90. Blood chemistry *will show a low sodium and low sugar,* a high potassium and a high urea and nonprotein nitrogen. The blood count shows secondary anemia, with a relative *lymphocytosis. Anorexia, nausea,* occasional vomiting and other signs of gastrointestinal disturbance are prominent symptoms. There is either a hypochlorhydria or achlorhydria. Loss of weight and various nervous *symptoms* are common. The sexual function is greatly diminished; the heart is weak, and the B-M-R is somewhat below normal.



Fig. 23—Adrenal cortical tumor with pituitary basophilic adenoma in a woman Age 36 years. (Philadelphia General Hospital.)

Tumors of the Adrenal Cortex: Adrenal cortex tumors may be congeni-

The Pancreas (Islands of Langerhans)

The endocrine portion of the pancreas resides in the islands of Langerhans.

Anatomy and Physiology of the Islands of Langerhans

Anatomy: The islands of Langerhans are found between the alveoli of the pancreatic structures, and are more than twice as numerous in the tail as in the head of the pancreas. They are composed of small groups of polyhedral cells forming a network in which many capillaries ramify. The islands of Langerhans are made up of three types of cells which have different staining qualities. The beta cells, which are the most numerous, secrete the hormone, insulin.

Hormone: Several principles said to possess a blood pressure lowering action have been extracted from pancreatic tissue devoid of the islands. These are questionable hormones. The actual hormone secreted by the islands of Langerhans is insulin.

Physiologic Action of Insulin: Insulin controls carbohydrate metabolism by enabling the tissues to burn sugar, by increasing the ability of the liver and muscles to store sugar in the form of glycogen, and by inhibiting the formation of sugar, amino acids and perhaps fat in the liver. It thus regulates the amount of glucose in the circulating blood, and the amount of glycogen stored in the liver and the muscles as a ready source of energy. The islands of Langerhans are said to be influenced by the pancreatropic and contrainsulin principles of the anterior pituitary body.

Diseases of Islands of Langerhans Origin

Hyperactivity of the islands of Langerhans causes an increased secretion of insulin and therefore hypoglycemia.

Hypoactivity of the islands of Langerhans causes a scarcity of insulin, therefore hyperglycemia.

Hypoglycemia or Glycopenia

The normal blood sugar after a 12 hour fast is between 90 and 120 mg. per 100 cc. of blood. Values less than 70 mg. are considered as hypoglycemia. Hypoglycemia or an abnormally low sugar content of the blood may be caused by an overdose of insulin; by adenoma or other tumor in the pancreas which stimulates the islands of Langerhans to greater activity; by hypertrophy or hyperplasia of the islands, and by hypoactivity of the pituitary, adrenals and thyroid. Hypoglycemia also occurs in diseases of the liver in which there is diminished storage or increased release of glycogen, after severe muscular exertion, and in conditions in which sugar is rapidly lost from the body. Hypoglycemia with excessive storage of glycogen in the liver and infantilism is known as Von Gierke's disease.

Symptoms of Hypoglycemia. These depend upon the degree of blood sugar impoverishment. *In moderate hypoglycemia* there is gnawing hunger, marked weakness and fatigue, sweating, anxiety, irritability, restlessness and nervous trembling. These symptoms may come on suddenly or may be more or less constant. They are relieved by taking sugar or by frequent feeding. Marked hypoglycemia may come on suddenly with severe sweats, cold clammy skin, stupor, amnesia or coma; there may also be muscular twitchings, local or general convulsions and absent or weak deep reflexes. The timely administra-

Ephedrine and synephrin possess an adrenalinlike action and are as active by mouth as they are parenterally. These are also active when applied to mucous and serous surfaces.

Diseases of the Adrenal Medulla

Hyperfunction of the adrenal medulla, as is found in certain tumors, hypertrophy and hyperemia of that gland, may cause vascular hypertension with or without sclerosis, arteriosclerosis, and hyperactivity of the sympathetic division of the autonomic nervous system and may also cause hyperglycemia.

Hypofunction of the adrenal medulla may probably in part be responsible for asthenia, hypotension, muscular insufficiency and hypoglycemia.

Adrenal Tumors: Tumors of the adrenal medulla are of three types:

(1) *Neuroblastoma:* These are found in infants and young children The tumor is not large; it usually affects the right adrenal and metastasizes to the liver, which becomes enormously enlarged and to the mesenteric lymph nodes One variety of this type metastasizes to the orbit, to other parts of the skull, the ribs, sternum, long bones, and occasionally the internal organs

(2) *Ganglioneuroma:* These are found in children and young adults They may be comparatively benign and may cause hypertension, hyperglycemia and symptoms attributable to hyperstimulation of the sympathetic nervous system

(3) *Pheochromocytoma* (Chromaffin Cell Tumors, Paraganglioma): These are usually encapsulated benign tumors; they are found in old people and may not cause any symptoms. Occasionally a paraganglioma, like a malignant blastoma, may cause periodic intermittent attacks of hypertension. malaise, tachycardia, profuse sweating, headache and nervousness.

Neurocirculatory Asthenia (Autonomic Ataxia): This condition presents varied manifestations of instability of the autonomic nervous system. Crile attributes this syndrome to hyperfunction of the adrenal medulla and describes it as "excessive stimulation of the adrenal sympathetic nervous system"

Other Adrenal Lesions

Various lesions may affect one or both glands as a whole, or either or both cortices or medullae. These lesions may be various types of primary or secondary tumors, or abscesses; or they may be caused by tuberculosis, miliary or caseous; by syphilis of various types and stages; and also by hemorrhage, inflammations, hypertrophy, atrophy, and degenerations.

The symptoms of these lesions depend upon whether they are stimulating or destructive and whether they affect one or both glands, or the cortex or the medulla of either gland, as well as upon the amount of damage done by them Cysts, if large, may destroy the adrenals and cause renal pressure symptoms Hemorrhage, when large, will cause sudden death. Syphilis and tuberculosis may cause Addison's disease or hypocortical asthenia

Waterhouse-Friderichsen Syndrome: This is an acute fulminating fatal condition, characterized by high fever, weak rapid pulse, intense cyanosis, and purpuric hemorrhages into the skin. Nausea, vomiting, and various signs of meningitis are always present. Among the postmortem findings is massive bilateral adrenal hemorrhage.

medicinal purposes are plain insulin, protamine zinc insulin, and crystalline insulin.

Ketosis or diabetic coma may occur in diabetics and should be differentiated from insulin shock or hypoglycemia.

Differential Table of Coma in Hypoglycemia and Hyperglycemia

Hypoglycemia or Hyperinsulinism or Insulin Shock Coma	*Hyperglycemia or Hypoinsulinism or Diabetic Coma (Ketosis)*
1. Prodromal Symptoms	1 Prodromal Symptoms:
(*a*) Sudden onset with rapid manifestation of prodromal symptoms	(*a*) Gradual onset, prodromal symptoms of varying types.
(*b*) Coma may be preceded by sudden weakness, hunger pain, sweating, double vision, great anxiety, nervous trembling, delirium, convulsions and coma.	(*b*) Coma may be preceded by a cyanotic dyspnea, nausea and vomiting, anorexia, thirst, abdominal cramps and constipation. There also occur marked headache with weakness, malaise, muscular flaccidity and general irritability, restlessness, progressive sleepiness followed by stupor and coma.
2. During the State of Coma:	2 During the State of Coma
(*a*) Breathing is rapid and shallow	(*a*) Breathing is slow, deep and sighing (Kussmaul's type of air hunger respiration).
(*b*) Appears as if asleep.	(*b*) Patient appears ill.
(*c*) No characteristic odor on the breath.	(*c*) Fruity odor on the breath, cherry red lips, and flushed cheeks.
(*d*) Unconsciousness, though plantar reflexes are elicitable and convulsions often occur	(*d*) Complete unconsciousness with an absence of reflexes and only occasionally convulsions
(*e*) Eyeballs not soft.	(*e*) Soft eyeballs (Riesman's sign).
(*f*) Profuse sweating (a constant and characteristic sign)	(*f*) Marked dehydration, no sweating
(*g*) Low blood pressure	(*g*) Low blood pressure though at times it may be high
(*h*) Subnormal temperature	(*h*) Hyperpyrexia common.
Laboratory Findings	*Laboratory Findings*
(*i*) Hypoglycemia marked.	(*i*) Hyperglycemia usually marked.
(*j*) Absence of glycosuria.	(*j*) Glycosuria and acetonuria are usually present.
(*k*) No leukocytosis	(*k*) Leukocytosis with normal differential count
(*l*) Carbon dioxide alveolar air content within normal limits.	(*l*) Alveolar air carbon dioxide content greatly reduced
(*m*) If due to an overdose of insulin, it is seldom fatal when properly and promptly treated. If due to a tumor or other lesion of the pancreas, repeated attacks may eventually prove fatal.	(*m*) Slow response to medication, at times fatal

tion of glucose will usually produce spontaneous recovery.

Hyperglycemia

An increase in the sugar content of the blood above the normal is usually caused by a hypoactivity of the islands of Langerhans in which an insufficient amount of insulin is produced, or in conditions where the tissues are incapable of utilizing sugar at the normal rate. Hyperglycemia occurs in diabetes mellitus, in bronzed diabetes (hemachromatosis), and it may also occur in certain brain diseases or tumors, skull injuries, meningitis, hyperthyroidism, hyperadrenalism, hyperpituitarism, and in increased hydrogen ion concentration of the blood.

Diabetes Mellitus: This is characterized by hyperglycemia, glycosuria, polyuria, increased appetite and thirst, and loss of weight. Other symptoms such as pruritis, skin lesions, neuritic pain and visual disturbances are frequently encountered. Complications such as carbuncles, furuncles, ulceration and, at times, gangene of an extremity and arteriosclerosis, coronary disease, ketosis, and diabetic coma may occur in untreated cases. Diabetes mellitus is often a familial disease and occurs more frequently among the obese than in the nonobese. It may occur during childhood or during adulthood. The disease is of insidious onset and may not be suspected by the patient until severe symptoms develop. The diagnosis of diabetes mellitus is based on the presence of glucose in the urine, an abnormal amount of sugar in the fasting blood and the glucose tolerance test. The glucose tolerance test will show a high curve which indicates a low sugar tolerance. (For the significance of glycosuria, hyperglycemia and sugar tolerance, see p. 1012).

Treatment: In the treatment of diabetes mellitus it is important to adjust the patient's diet to his capacity to utilize a sufficient amount of carbohydrates without causing a hyperglycemia, the amount of fats without causing acidosis, and the proper amount of protein required for the individual's need. Should the patient be unable to utilize the minimum requirement of carbohydrates without causing hyperglycemia then a sufficient number of units of insulin is to be injected subcutaneously about one-half hour before each meal. The injected insulin will thus substitute for the insulin scarcity caused by the hypoactivity of the islands of Langerhans.

In order to determine the amount of carbohydrates, fats and proteins required by the individual, the number of Calories necessary for his basal maintenance must first be calculated. Each kilogram of body weight requires about 30 Calories. A patient weighing 60 kilograms would require 1800 Calories in 24 hours, which under certain circumstances may be divided as follows—carbohydrates 360 Calories, proteins 240 Calories, and fats 1200 Calories. One gram of carbohydrates yields 4 Calories, therefore 90 grams of carbohydrates; 1 gram of proteins yields 4 Calories, therefore 60 grams of proteins,; 1 gram of fats yields 9 Calories, therefore 133.3 grams of fats.

These rates may have to be readjusted under various circumstances. In addition to the Caloric requirements there must be added to the diet salts, vitamins and fluids. When insulin is necessary it is well to bear in mind that 1 unit of insulin will take care of about 2 5 Gm. of glucose. The varieties of insulin used for

medicinal purposes are plain insulin, protamine zinc insulin, and crystalline insulin.

Ketosis or diabetic coma may occur in diabetics and should be differentiated from insulin shock or hypoglycemia.

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(*m*) If due to an overdose of insulin, it is seldom fatal when properly and promptly treated If due to a tumor or other lesion of the pancreas, repeated attacks may eventually prove fatal.	(*m*) Slow response to medication, at times fatal

The Gonads (Male and Female)

The gonads or sex glands are the organs which primarily determine the sex of the individual and make reproduction possible. The reproductive function of the gonads is under the control of the gonadotropic hormone of the anterior pituitary body. Prolan A is believed to stimulate spermatogenesis in the male and follicle ripening in the female. Prolan B is said to stimulate the production of the male hormone secreted by the interstitial cells and the lutein hormone secreted by the corpus luteum.

The Male Gonads

From the endocrinologic viewpoint the testes are the most important structure of the male gonads They have both an external and internal secretion.

Anatomy: The adult testes vary somewhat in size in different individuals; they measure approximately 4 by 2 5 by 3 cm, each weighing from 10 to 14 Gm They are suspended in the scrotum; the left usually hangs a little lower than the right Each testicle is covered by three coats, *viz*, the tunica vaginalis testes, the tunica albuginea, and the tunica vasculosa. Structurally the testis is divided into numerous lobules by offshoots from the tunica albuginea The lobules contain the convoluted seminiferous tubules Between the seminiferous tubules there is a stoma of connective tissue which harbors the *interstitial cells of Leydig* The seminiferous tubules are lined by the spermatogonia cells from which, by a complicated process, the *spermatozoa* are developed. The spermatozoa and the fluid element of the semen are an external secretion and not a hormone It is, however, believed that the spermatic cells also produce a hormone though as yet not identified Spermatogenesis begins at puberty and continues to old age.

Hormone: The testicular hormone, *testosterone* (male hormone), is secreted by the interstitial cells of Leydig. A similar hormone with slight modification of its formula is recoverable from male urine and is known as *androsterone* Testosterone is now being manufactured synthetically and is obtainable as testosterone propionate, or by various trade names such as Oreton, Perandren, Androstene B, etc.

The other testicular substance is believed to be derived from the germinal epithelium, probably from the cells of Sertoli, and is named *inhibin* It is supposed to inhibit the anterior pituitary gonadotropic hormone, therefore causing testicular atrophy.

Function of Testosterone: Testosterone assists in the maturation of the skeleton, it accelerates epiphyseal ossification and helps the development of the skeletal muscles and the larynx. It is responsible for the male type of hair and fat distribution, and is concerned with the development of the male sex organs, sex function, and, to some extent, spermatogenesis. Testosterone propionate reduces benign hypertrophy of the prostate and may stimulate libido.

Pathology: The testicles may become injured by disease or trauma or they may be invaded by various types of tumors which may alter their function and cause hyper- or hypogenitalism. Endocrinopathies of testicular origin may be congenital or acquired, and may be primary or secondary, the latter being the result of disease of other endocrine glands such

as the pituitary, thyroid, suprarenal bodies, and probably pineal and thymus.

Endocrine Diseases of Male Gonad Origin

Cryptorchism: Cryptorchism (retained or undescended testicles) may be unilateral or bilateral.

Unilateral Cryptorchism: This may not be attended with pronounced hormonal disturbance since the one normally situated testicle may perform the required functions. These individuals, as a rule, show some sparsity of facial hair, and are somewhat hypogonad.

Bilateral Cryptorchism: This is always attended with aspermatogenesis, because the intraäbdominal temperature destroys the spermatogenic function Other manifestations vary Some of the adults may have normal male secondary sex characteristics, be of good stature, and have fairly normal male hair distribution, others may be markedly lacking in secondary male sex characteristics The external genitalia are poorly developed; the hair distribution is of the female type, and the breast development may resemble the female type

Hypogonadism (Eunuchoidism): Hypogonadism may be of various types.

Primary Hypogonadism: This presents the following characteristics: The trunk is short, the upper and lower extremities are disproportionately long, the face is small and beardless; the genitalia are small or rudimentary, and the voice is high pitched.

Pituitary Hypogonadism: This is characterized by a comparatively long trunk and proportionately shorter lower extremities The face is rounded, the skin is pale, and facial hair is scarce There is the usual hypopituitary fat distribution with large pads of fat over the trochanteric region, the breasts may be prominent, the genitalia are pooorly developed and the prostate is small Aspermatogenesis is the rule.

Thymus Type of Hypogonadism: In this condition the body length is somewhat shorter than the lower extremities. The upper and lower extremities are cylindrical and well molded; the skin is of soft texture; the upper lateral incisors are rudimentary; the beard is sparse, the pubic hair is of the female type, the genitalia may be rudimentary

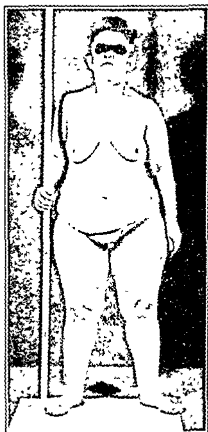


Fig 24 — Pseudohermaphroditism Age 42 years, with bilateral cryptorchism Note the well developed breasts, female hair distribution on the mons, no hair upon the face and female-shaped pelvis

or of nearly normal size, libido and potentiality are subnormal; sterility occurs in a large proportion of cases, and homosexualism is fairly common

Hypothyroid Type: This type shows evidence of cretinism or of myxedema, the body is thick; hair distribution is scanty, the skin is lustreless and me-

Eunuchism or agonadism is acquired after castration The characteristics depend upon the age at which the individual was castrated. Castration during *early childhood* prevents the development of sexual maturity and function and of *secondary sex characteristics. Castration after puberty* causes retrogression

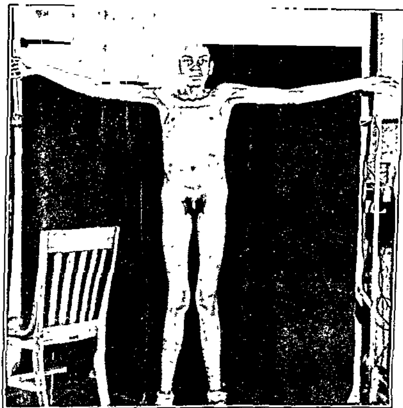


Fig 25—Congenital eunuchoidism Age 24 years Note the length of extremities in proportion to the trunk (Philadelphia General Hospital)

lastic; the stature is undersized, and the genitalia are subnormal in development and function

Eunuchoidism: This is congenital hypogonadism of a severe degree and may show characteristics of any one of *the other types or of all the types combined.* These individuals are always sterile and devoid of both libido and potentiality.

of secondary sex characteristics, though the penis remains normal size. Eunuchs are sterile but are not entirely *devoid* of libido and potentiality. In body *development the eunuch may be fat or thin*

Pseudohermaphroditism: Pseudohermaphroditism is characterized by the presence of characteristics of both sexes. The predominately male individual may have breasts and deformity of his gonads

somewhat resembling the female genitalia and the general appearance and mannerisms may be feminine.

Hypergonadism: Hypergonadism may occur during childhood or during adulthood. *Childhood hypergonadism* or *macrogenitosomia precox* may occur at an early age and is often caused by an adenoma of the pineal body, the pituitary gland or the adrenals The child so affected develops premature masculinity

Adult hypergonadism is characterized chiefly by increased function of the sex glands (SEE ALSO: p. 707).

The Female Gonads

The ovaries possess *two internal and* one external secretions. The internal secretions are: (*a*) The follicular hormone and, (*b*) the luteal hormone, and the external secretion is the production of the ovae. Both of these secretions are under the control of the gonadotropic hormones of the anterior pituitary body

Anatomy of the Ovaries: The ovaries are two nodular *grayish-pink* bodies situated one on either side of the uterus attached to its broad ligament behind and below the Fallopian tubes Each ovary measures approximately 4 by 2 by 1 cm., and weighs from 2 to 3 5 Gm, and is divided into a cortex and medulla. The entire gland is enveloped by a layer of germinal epithelium, the germinal epithelium of Waldeyer, which is the source of the ova and follicles The cortex is composed of interstitial connective tissue in which are embedded the follicles *which later harbor the ova* The medulla is made up of connective tissue, smooth muscle and elastic fibers, blood vessels and lymphatics At birth each ovary contains its *alloted number* of primordial follicles: no new ones are added throughout life From birth to puberty, before menstruation is established, *many of the primordial follicles* reach the stage of ripening and then retrogress and undergo atresia These atretic follicles elaborate the follicular hormone which in the prepuberty stage *influences the* characteristic somatic development of the girl (secondary female sex characteristics).

Beginning at puberty and continuing to the menopause, the ovum is *extruded from* the ovary *once a month* as each follicle ripens, and finds its way into the uterus The corpus luteum originates at the point in the ovary through which the *ovum has ruptured and begins its de*velopment If the ovum remains unfertilized, the corpus luteum continues to develop until after menstruation has taken place when it retrogresses and becomes absorbed Should the ovum become fertilized, then the corpus luteum continues to grow and elaborates its hormone, which prepares the endometrium *for the reception of the ovum* and aids in the maintenance of pregnancy The corpus luteum continues to grow within the ovary, reaching its maximum at the 13th week of pregnancy when the *placenta is formed and it remains at* that size until the end of pregnancy, when it becomes absorbed

The placenta when fully formed elaborates *the follicular and the pituitary*-like hormone such as is found in the blood and urine of pregnancy, namely, Antuitrin S, A P L, Follutin, etc.

The Ovarian Hormones: The follicular hormone which is variously known as Estrin, Estradiol, Theelin, Theelol, Folliculin, Progynon, Amniotin, etc., is produced in the graafian follicle and in the placenta A *small quantity* of this hormone is found in the blood and urine of normal adult women from puberty to

the *menopause*. The quantity is appreciably increased after menstruation. A great abundance of this hormone is found during pregnancy and in the presence of certain ovarian tumors.

Function of the Follicular Hormone: It causes development of the secondary sex characteristics of the female, development of menstruation, growth of the myometrium, development of the endometrium, and of the adult type of vaginal mucosa. It is also responsible for the development of the mammary ducts, and for rhythmic uterine contractions.

Function of the Luteal Hormone: This hormone is known as progesteron It helps to prepare the endometrium for the reception of the ovum and inhibits ovulation, menstruation, and uterine contractions; it maintains pregnancy, and causes the development of acinar tissue in the breasts.

Menstruation: This is a characteristic monthly function of normal nonpregnant adult women which begins at puberty and stops at the menopause. The normal cycle is initiated at puberty by the follicle maturing and the luteinizing hormones of the anterior pituitary body acting upon the follicular and luteal hormones of the ovary. About 12 or 15 days after the ovum is released from the ovary, if not fertilized, it is cast off from the uterus, together with endometrium tissue, mucus, degenerated epithelium and blood from the ruptured premenstrual endometrial blood vessels. This constitutes the menstrual flow, which generally occurs every 28 days and lasts from four to six days.

Pathology: Ovarian function may be disturbed by disease of the ovaries and by pathologic lesions in the anterior pituitary body, the adrenal cortex, the thyroid, and, possibly, by the pineal body and the thymus.

Endocrine Diseases of Ovarian Origin

Cysts and Tumors: Lesions, such as cysts and tumors, either congenital or acquired, may affect the internal secretion of the ovary and produce hyperfunction, hypofunction or afunction of these glands.



Fig. 26—Pubertas precox. Age seven years Due to a granulosa tumor of the ovary.

Granulosa Cell Tumors and Theca Cell Tumors: These stimulate the production of large amounts of the estrogenic hormone, therefore in the young they may produce premature matronism (premature puberty) with early menstruation and precocious premature secondary sex characteristics. In older women they may produce metrorrhagia, and in those past the menopause, there

may be reinstituted periodic menstrual bleeding without ovulation.

Arrhenoblastomata: This may cause masculine characteristics and such changes as amenorrhea, mammary atrophy, hypertrophy of the clitoris and hypertrichosis upon the face and body. Removal of such a tumor causes the disappearance of the male characteristics and the return of femininity

Hypoovarianism: Hypoövarianism may cause *amenorrhea, hypomenorrhea,* dysmenorrhea and sterility, as well as somatic changes, the severity of which *depend upon the degree of hypofunction* and the age at which it started

Preadolescent Hypoövarianism is *characterized by failure in the development* of the breasts and genitals and failure also in the initiation of menstruation at puberty. The individual is usually *tall;* the lower extremities are proportionately longer than the trunk, and there is a tendency to obesity

Adolescent Hypogonadism is characterized by incomplete development of secondary sex characteristics Menstruation is delayed, *and when it does appear* is scanty, irregular and may be painful The somatic development is of two types · The one is the thin type, *slender and tall* with long extremities and long slender fingers and toes, and narrow chest with *immature breasts; the other is the obese,* pudgy type with large breasts and trochanteric pads of fat. Both types are usually sterile.

Adult Hypogonadism, or late castrates, present amenorrhea, vasomotor phenomena such as "hot flushes," chills, sweats, and parasthesia with functional nervous phenomena The individuals may become fat or stay *thin,* and a growth of long coarse hairs upon the chin and around the corners of the mouth appears

Hypogonadism in both sexes may occur in association with infantilism and is due to hypofunction of the anterior pituitary body. The hypogonadism associated with cretinism is due to hypothyroidism.

Hyperovarianism; Pubertas Precox: In the preadolescent stage, hyperovarianism or pubertas precox is characterized by *signs of early maturity, i. e.* early appearance of pubic hair, marked enlargement of the breasts, precocious *development of the external genitalia,* and early initiation of menstruation There is also a rapid skeletal growth *during the first decade but growth stops* early in the second decade because of premature epiphyseal ossification

Adult Hyperovarianism: This may present various manifestations such as increased libido, nymphomania, unusual *fertility and, in some cases, metrorrhagia,* uterine hypertrophy and other signs of hyperfemininity.

Virilism: This term is generally applied to women who present masculine characteristics in mannerism, hair *distribution, and muscle development. At* an early stage they show evidence of hypersexuality and later there occurs *sexual reversion. Examples of this type* are seen in pituitary basophilism, adrenal cortex tumors and ovarian arrhenoblastoma

For nonendocrine diseases of the female genitalia, see p 695

Puberty, Menstruation, Adolescence, and Menopause

Puberty (Female): Puberty is the transitional period during which the child of either sex develops definite sex characteristics In the female this period usu-

ally occupies about two years or longer, beginning about one year before the appearance of the first menstrual flow and lasting until the secondary sex characteristics are well established.

Physical Changes: During the year before the appearance of menstruation, there is a decided increase in body growth, the rate of growth being greater during that year than during any one preceding or subsequent year. The pelvis becomes wider and more flaring, fat gradually covers the shoulders, thorax, hips, and pubes. The breasts develop their duct systems and assume a spherical shape with the areolas and nipples at their apices As the breasts develop, hair begins to grow upon the mons veneris which becomes well covered by the time the first menstruation appears. Axillary hair usually appears later.

The pituitary body, the thyroid, the ovaries, and the adrenals continue their development for some time after the first menstrual epoch, as do also the uterus and the external genitalia The thymus gland and the pineal body retrogress

Psychic Manifestations: The transformation of the carefree child into a sexually conscious being in whom the endocrines, the nervous system, and the general development are still in a state of imbalance, is bound to cause many new and unpleasant sensations. Such sensations in a person possessing a labile nervous system are bound to be converted into somatic demonstrations. During the pubertal period, these children often show a decided change in both their psychic and somatic manifestations. They may be moody, secretive, stubborn, irresponsible, nervous, and irritable. They may often suffer from emotional dreams and restlessness, and also have fits of uncontrollable laughing and crying. Often these children suffer from cardiac palpitation, digestive disturbances, nausea, vomiting, and flatulence, and from such other nervous manifestations as headaches, sweating, flushing, and sudden pallor. Certain skin diseases, such as acne, urticaria, herpes, and other blemishes, often occur during this period and may be continued during adolescence.

The severity of the nervous manifestations depends largely upon the stability of the child's nervous system and her general environment. Many girls show few nervous manifestations and others are definitely unbalanced.

Premature Puberty (Pubertas Praecox, Premature Matronism): The premature appearance of adult bodily conformation and of menstruation or pseudomenstruation results from lesions of the endocrine glands associated with such development Premature bodily development without premature menstruation is found in those suffering from pituitary tumor or adrenal tumor. Premature menstruation occurs in those suffering from an ovarian tumor, while virilism may result from both pituitary and adrenal cortical tumors.

Delayed Puberty: This may be the result of an organic lesion or functional hypoactivity of the various endocrines The five types are as follows.

1. The hypothyroid type: This is associated with stunted growth and general immaturity.

2 The pituitary type: This is associated either with Froehlich's syndrome or with pituitary dwarfism

3. The primary gonad types· One type is fat, having fat pads over the trochanteric regions and poorly developed secondary sex characteristics; the other type is tall, thin, and has long arms and legs.

4. The Lilliputians: These have childlike configurations throughout their lives (*microsomia*).

5. Functional hypoactivity of several of the endocrines not associated with any organic lesion. These do not as a rule show any definite somatic characteristics.

Menstruation: The age at which the first menstrual flow (the menarche) appears varies among the normal girls of different climates, races, and types In the temperate zones, the menarche appears between the ages of twelve and fourteen years. Occasionally it may appear as early as ten and one-half years or it may be delayed beyond the sixteenth year. Menstruation and ovulation usually appear at a younger age in warm climates, among the heavy-set, broad-boned and dark-skinned girls, and it is delayed in cold climates, among the light-complexioned, and the tall, thin willowy girls

The appearance of the first menstrual flow indicates that puberty is well on its way, but it is not a sign of sexual maturity. Maturation may not be completed before the eighteenth or twentieth year

The Menstrual Cycle: There is no definite regularity following the first menstrual flow The intervals between bleedings may be delayed for many months or they may be shortened, occasionally there may be continuous bleeding for several weeks It may be a year or more before regular cycles are established. The first few menstrual periods may be accompanied by severe pain (dysmenorrhea) This pain may occur in some women with each period Other women experience no pain The normal menstrual cycle is usually counted as twenty-eight days, it comprises the interval between the first day on which bleeding occurs in one month and the first day of bleeding in the following month The bleeding time is usually between four and five days These figures are not definite, since so many women have their own peculiar cycle, varying between twenty-two and thirty-two days or longer The bleeding time and quantity of blood discharged also vary. Some women may bleed for two or three days, others for five or eight days, and the flow may be scanty or profuse However, when the cycle is once established in the normal woman, with the exception of the time of pregnancy and lactation, it is usually maintained with occasional variations of one or two days until the menopause is reached

Mechanism of Menstruation: The menstrual cycle is initiated by the follicle-maturing hormone and the luteinizing hormone of the anterior pituitary body which act upon the follicular and luteal hormones of the ovary About twelve to fifteen days after the ovum is released from the ovary, if it is not fertilized, it is cast off from the uterus, together with the blood from the ruptured, newly formed, premenstrual endometrial blood vessels, endometrial tissue, degenerated epithelium, and mucus This constitutes the menstrual flow or catamenia When the cyclic bleeding is associated with ovulation, it is known as "true menstruation", and when it occurs independent of ovulation, it is termed "pseudomenstruation"

Nomenclature of Menstrual Disorders:

1 Amenorrhea: absence of cyclic menstruation This may be physiologic or pathologic, primary or secondary.

2 Oligomenorrhea: abnormally infrequent or delayed menstruation

3 Hypomenorrhea: scanty menstruation.

4. Dysmenorrhea: painful menstruation.

5. Premature menstruation: precocious puberty (pubertas praecox).

6. Menorrhagia: prolonged menstrual flow.

7. Metrorrhagia: prolonged and irregular uterine bleeding, not necessarily connected with the menstrual cycle.

8. Pseudomenstruation: menstruation without ovulation.

9. Vicarious menstruation: cyclic extrauterine bleeding.

Adolescence (Youth, Teen-agers): Adolescence comprises the period between puberty and maturity. During this period, the body attains its full stature, the sex organs and their function, and the endocrine glands connected with sex function become fully developed, and the nervous system becomes stabilized The age of maturity varies among different races and in different climates. Those that reach puberty early, mature early In the temperate climate, maturity is usually reached between the ages of eighteen and twenty years.

The Period of Sexual Productivity: This is the reproduction period which begins at maturity and ends at the menopause.

The Climacteric (Female) and the Menopause: The terms "climacteric" and "menopause" are loosely considered as being synonymous However, literally, the term "climacteric" denotes the transitional period during which the woman changes from a potentially reproductive person to a nonproductive one; while "menopause" denotes the cessation of the menstrual function which is only one of the manifestations of the climacteric.

The climacteric usually begins several years before cyclic menstruation stops, and it usually continues for several years thereafter. The menopause, in the temperate climate, usually occurs any time between the forty-fifth and fiftieth year. It may, however, occur prior to the forty-fifth year or later than the fiftieth year.

The climacteric is the period during which there is a gradual regression of the productive organ; it is the antithesis of puberty during which the reproductive organs mature. During both periods, there occurs a definite hormonal imbalance which causes many psychic and somatic changes During the climacteric, there is a deficiency of ovarian hormones (estrone and progestrone) and a relative increase of the pituitary sex hormone (prolan). There are also definite evidences of disturbances of most of the endocrine glands

Symptomatology: The symptoms encountered during the climacteric vary; the severity depends largely upon the stability of the nervous system, the physical stamina, and the environment of the person The patient who had presented marked psychic disturbances at puberty usually suffers severe nervous manifestations during her menopause. The cessation of the menstrual flow varies in different women Some women stop menstruating abruptly Others stop slowly, the menstrual flow becoming less profuse and occurring at longer intervals Still others may develop menorrhagia or metrorrhagia. Occasionally a woman, after being amenorrheic for many months or years, may suddenly have a profuse menstrual flow. Such women should be carefully examined to exclude a pelvic neoplasm.

Ovulation usually stops with the cessation of menstruation. In many instances, ovulation stops and pseudomenstruation continues Occasionally ovulation may continue for some time after the

menstrual flow has stopped. This accounts for the occasional woman who becomes pregnant after her menstruation has ceased.

Among the earliest symptoms, often preceding the cessation of menstruation, is vasomotor instability, namely "hot flushes or flashes," chills, and sweats. These come on in paroxysms during the day or night. The attacks may come on at short or prolonged intervals and may last several minutes. Excitement intensifies the attacks. Accompanying these attacks, and often independent of them, there occur periods of weakness, depression, vertigo, anxiety, headaches, cardiac palpitation, nausea, vomiting, insomnia, joint and muscle pain, paresthesias, hyperesthesias, itching, and various psychic manifestations, such as crying spells, mental depressions, melancholia, and occasionally some form of psychosis Any preexisting neuropsychiatric tendencies are aggravated during the climacterium. Many women approach the climacterium and pass beyond it with very little or no perceptible discomfort.

Various somatic changes develop gradually after the menopause which are not entirely due to aging The epithelial elements of the breasts are gradually replaced by fibrous tissue, and in thin women the breasts shrink and the fat distribution becomes altered. The axillary and pubic hair becomes scant Hair may appear upon the face The primary and secondary sex organs gradually atrophy. The skin may develop various lesions, such as keratosis, pigmentation, papillomata, and other dermatoses The accumulation of fat, particularly over the abdomen, or generalized obesity is not uncommon during the menopause or the postmenopausal era.

SECTION 13

The Nervous System

CHAPTER XXVII

Anatomy, Physiology and Examination of the Nervous System

The nervous system is composed of specialized cells and their projecting fibers (the neuron) whose function it is to guide the destinies of the individual in relation to his own vital processes and to his surroundings. It may be considered the ordinance department of the body which, by virtue of its elaborate telegraph and decoding system, perceives, transmits and decodes all types of sensory impulses from the various tissues and organs of the body and finally delivers suitable motor, secretory or other responses to these impulses to their proper destinations

The neurons are held together and supported by neuroglia, which are a special type of cells also of ectodermal origin, but which do not participate in conduction or transmission of impulses.

The nervous system is divided into three parts · (1) The central or cerebrospinal nervous system, (2) the peripheral nervous system, and (3) the autonomic or vegetative nervous system.

(1) The Central, Somatic or Cerebrospinal Nervous System: This system includes the brain, which is encased in the cranium, and the spinal cord, a continuation of the brain, which is contained within the spinal column

(2) The Peripheral Nervous System: This is made up of a series of nerves through which both the brain and the spinal cord exert their influence upon the various structures and functions of the body. The nerves contain sensory and motor fibers. The cerebrospinal, central or somatic nervous system controls the voluntary movements of the body, and the general and the special senses.

(3) The Autonomic or Involuntary Nervous System: The autonomic nervous system presides over the functions of the body not under voluntary control, *i. e.*, the heart, lungs, abdominal viscera, the blood vessels, the secretory and excretory glands, etc. The autonomic system is divided into the sympathetic and parasympathetic divisions. The parasympathetic or craniosacral autonomic system contains fibers from the brain and the spinal cord which approach the peripheral ganglia through the 3rd, 7th, 9th, 10th and 11th cranial nerves, and through the pelvic nerve from the 2nd, 3rd and 4th sacral nerves.

The sympathetic fibers consist of a paired trunk of nerve fibers and ganglia extending from the superior cervical ganglion to the ganglion impar anterior to the 5th sacral vertebra (SEE p 825).

Anatomy and Physiology of the Nervous System

The Central or Cerebrospinal Nervous System

The Neuron

The unit of the entire nervous system is the nerve cell or neuron The nerve cell or neuron consists of a mass of protoplasm in the center of which resides a nucleus and from its periphery spring two types of elongated processes or fibers, known as dendrites and axons. *The dendrites* are short fibers, irregular in shape, having many branches and

terminating a short distance from their cell body. Each neuron usually possesses several dendrites, though in some neurons they are absent. *The axon* or axis cylinder is usually single, of small diameter, smooth and of relatively great length, terminating in numerous fine branches at some distance from its cell origin. The dendrites and axons form the nerve fibers. A large number of *nerve fibers (from a large number of* cells) bound together in a universal sheath forms a *nerve trunk. Impulses* arising in a cell are transmitted by its axon to another cell.

The entire nervous system is thus composed of individual neurons (nerve cells and their tentacles) grouped in special types of bundles One type conducts impulses from the periphery to the central nervous system (*centripetal*); they form the sensory or afferent paths Another type conducts impulses from the central nervous system to the peripheral organs and muscles (*centrifugal*) and form motor or efferent paths.

Two other types run between the motor and sensory paths These are the important connecting links which form the intracentral tracts and are known as the *association conduction and reflex conduction*

The junction by which the impulse is transmitted from one cell to another is known as a *synapse*.

A ganglion is a collection or a mass of cells of similar function which serves as an energizing center for their nerve fibers. There are many ganglia distributed throughout the nervous system. Some are large, containing countless cells; others are small, being made up of a few cells. They may possess sensory, motor or special function.

The Nerve Fibers

The nerve fibers, both the myelinated and the unmyelinated, are the axis cylinder *processes of the nerve cells*. They are the chief components of the white substance of the nervous system and also, to some extent, help to form the gray matter. Through the nerve fibers relations are established between cells that may be either in close proximity or a great distance. The nerve fibers receive their nutrition and specific functions from their individual nerve cells; when detached from their cells they lose their ability to conduct impulses.

Normally the nerve impulse is conducted along the entire length of the nerve with undiminished intensity. When poisoned with a narcotic, the impulse is either diminished in intensity or abolished in the poisoned area

Degeneration and Regeneration: When an axon is severed the peripheral portion degenerates completely, while the central stump and the cell body show transitory changes.

Wallerian degeneration is that process when the distal (peripheral) portion of a cut nerve undergoes a chemical change with eventual complete disappearance of that portion of the fiber. The neurilemma becomes a chain of sheath cells.

Retrograde degeneration is that process where the central stump degenerates back to a node of Ranvier. The cell body shows the morphologic characteristic of the so-called axonal chromatolysis. The closer to the cell body the degenerative change, the more severe is this process.

Regeneration takes place only in the peripheral nervous system. The chain of sheath cells forms a pathway along which the new axon grows as a bud from the

central stump. The neurilemma is later re-formed from the sheath cell chain. Central neurones have no sheath cells and do not regenerate.

Nerve Trunks

The nerves are trunks containing many nerve fibers which are encased in a common sheath. The thickness of the nerve depends upon the number of nerve fibers it contains. As the nerve runs along its course, from its point of origin to its destination, it gives off many branches and individual fibers which innervate the various structures of the body. Some of the nerves carry only sensory fibers, others carry only motor fibers and still others carry both sensory and motor fibers. These last are known as mixed nerves. There are also nerves which carry special impulses to specialized organs such as sight, hearing, pain, touch, smell, secretion and other functions. The large nerves originate from or are attached to the brain, the spinal cord and some of the large ganglia. The brain has 12 pairs of nerves spoken of as the *Cranial Nerves*, and the spinal cord has 31 pairs of nerves spoken of as the *Spinal Nerves*. These nerves run in pairs so that each lateral half of the body is supplied by an identical nerve.

The Plexuses

A nerve plexus is a tangle of nerves made up of communicating branches of neighboring nerves or of the primary branches of nerve trunks. The nerves emanating from a plexus usually carry funiculi and primary fibers of several nerve trunks. Both the central and the automatic nervous systems possess many large and small plexuses.

The Cerebrospinal Fluid[1]

The cerebrospinal fluid is a specialized clear tissue fluid normally containing about 0.02 per cent of protein, 0.08 per cent of glucose, 0.73 to 0.75 per cent of chlorides, and a few lymphocytes. The spinal fluid pressure within the spinal canal is about 10 mm. of mercury or 200 mm. of water. In disease of the brain and meninges and in various infections, the spinal fluid will show changes in its color, composition and quantity (pressure) and may harbor various bacteria and yield specific reactions. The spinal fluid occupies the subarachnoid space, the various cisternae, the sheaths of the spinal and cranial nerves, particularly of the optic and auditory nerves, the ventricles of the brain and the spinal canal.

Function of the Cerebrospinal Fluid: It serves as a medium for nutrient exchanges in the nervous system, acts as a fluid buffer and helps to regulate intracranial pressure by increasing in quantity when the brain shrinks and decreasing in quantity when the brain expands.

Pathologically, when the intracranial pressure becomes excessive, as in brain tumor, there may result venous compression, papilledema, medullary anemia due to cerebellar wedging into the foramen magnum, and hydrocephalus.

The Encephalon (The Brain)

The brain encased in the cranium is composed of several parts that vary in structure and in function. It is composed of two identical lateral halves bridged together by an isthmus (corpus colosum), in which many fibers cross from one side of the brain to the other. The brain as a whole receives and trans-

[1] See p. 1023

terminating a short distance from their cell body. Each neuron usually possesses several dendrites, though in some neurons they are absent. *The axon* or axis cylinder is usually single, of small diameter, smooth and of relatively great length, terminating in numerous fine branches at some distance from its cell origin. The dendrites and axons form the nerve fibers. A large number of nerve fibers (from a large number of cells) bound together in a universal sheath forms a nerve trunk Impulses arising in a cell are transmitted by its axon to another cell.

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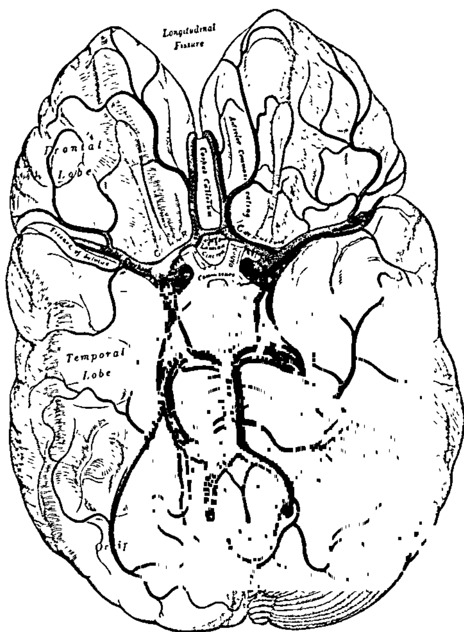
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THE BASE OF THE BRAIN

removed It will be noticed

1

that the . - from each other, this makes the ... longer than it really is.

mits impulses by way of the spinal cord and cranial nerves, and presides over most of the individual's functions.

External Appearance of the Brain: The shape of the brain usually conforms to the contour of the cranial cavity. Its upper surface is arched and its lower one flattened The gray matter is distributed over the periphery of the brain, giving it that grayish appearance; the white matter is situated internally. This is just the reverse of what is found in the spinal cord, where the white matter is external and the gray matter internal. The brain as a whole is surrounded by the three layers of meninges, namely, the *pia*, the *arachnoid* and the *dura*. It is well supplied with blood vessels, and with spaces for the housing of the cerebrospinal fluid. The *weight* of the brain varies with sex, age, and size of the individual. Its average weight in young adult men of medium stature is 1360 Gm. It is less in women and in persons of small stature or advanced age.

The Component Parts of the Brain: The brain may be divided horizontally into two planes, a higher and a lower plane.

The Higher Plane: This is represented by the cerebral hemispheres, each being divided into the frontal, parietal, temporal and occipital lobes. The cerebral hemispheres are ovoid in shape. They are separated from each other by the longitudinal fissure. The *corpus callosum* is a broad commissural band joining the two hemispheres at their undersurface. The cerebral cortex is concerned with intellectual, motor, sensory and special sense activity.

The Lower Plane: The base of the brain lies between the cerebral hemispheres and the spinal cord; it presents the medulla oblongata, the pons, the cerebellum, the cerebral peduncles, the optic tract, the optic chiasm and the optic nerves, the substantia perforata posterior, the *mammillary bodies*, the tuber cinereum, the pituitary body and the anterior perforated substance

The brain may also be divided longitudinally into three parts (1) The prosencephalon, or forebrain; (2) the mesencephalon or midbrain; (3) the metencephalon or rhombencephalon, the hindbrain or brain stem. Each of the three parts possesses varied structures that are important centers

The Brain Ventricles: There are four brain ventricles, which intercommunicate. They are situated one in each lateral hemisphere, the third lies between the two lateral halves of the diencephalon and the fourth in the rhombencephalon The central canal of the spinal cord, which is continuous in the medulla, opens into the fourth ventricle, which is continuous with the cerebral aqueduct and which in turn opens into the third ventricle. Near the anterior border of the third ventricle is situated a small opening, one in each lateral wall; this is known as the foramen of Monro or the interventricular foramen. It leads into the ventricle of each lateral hemisphere, the two lateral ventricles.

The Motor Pathways of the Brain and Cerebral Localization: The motor impulses from the cerebral cortex, which exercise voluntary control over the skeletal muscles, are conducted by way of the *pyramidal tracts*. This motor pathway, originating in the cerebral cortex and descending through the spinal cord, receives impulses from several sources, *i. e*, from the corpora quadrigemina through the tectospinal tract, from the vestibular nucleus by way of the vestibulospinal tract, from the large

motor cells of the reticular formation through the reticulospinal path, from the cerebellum, from the corpus striatum and possibly also from the thalamus or subthalamus by way of the thalamospinal tract. It is believed that motor impulses nerves or to the anterior gray columns of the spinal cord; and *lower motor neurons* or primary motor neurons which relay these impulses from there to the muscles. A third and much shorter conduction chain may be interposed be-

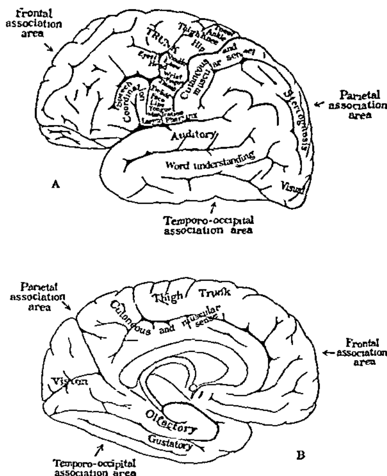


Fig 1—Side view of cerebrum showing specialized areas of cortex and their function. (From Morris' Anatomy, P Blakiston's Son & Co.)

may also be transmitted by way of the extra-pyramidal motor tract.

The pyramidal system consists of two motor unit chains: *Upper motor neurons* which conduct motor impulses from the cortex to the motor nuclei of the cranial tween the upper and lower motor units.

The Sensory Pathways: The sensory impulses that arise through the body are transmitted either to the cerebral cortex by various paths by way of the thalamus or are taken care of by

The pathway from the labyrinth transmits to the cerebellum the excitations that are produced in the semicircular canals by the pressure of the endolymph on the peripheral terminations of the vestibular nerve. The vestibular nerve leads to Deiters's nucleus and from this a pathway goes to the cerebellum.

The cerebrum influences cerebellar activity through corticopontocerebellar fibers.

Centrifugal Pathways: 1. The rubrospinal pathway consisting of three neuron systems The cerebelloölivary; the olivorubric, and the rubrospinal. The tract is direct in consequence of the double decussation. at first of the olivorubric system in Wernick's decussation, and then of the rubrospinal neuron in Forel's decussation.

2. The vestibulospinal pathway.

3. Ways of communication between Deiters's nucleus and the nuclei of the eye muscles Of these, the known paths are those from the third nerve nucleus of the opposite side and the sixth nerve nucleus of the same side

For Disturbances of Equilibrium and Orientation, see p 849.

The Pons (Pons Varioli)

The pons is continuous with the medulla oblongata. The peripheral neurons of the sixth and seventh, as well as the motor division of the fifth cranial nerves, originate in its gray matter It also contains the sensory nucleus of the fifth nerve and motor and sensory tracts which pass from the cord to the cerebellum and the cerebral cortex

The Medulla Oblongata (Spinal Bulb)

The medulla oblongata extends from the spinal cord at the level of the upper border of the atlas to the lower margin of the pons. The external surface of the medulla somewhat resembles the spinal cord except that it is considerably thicker The internal appearance and the distribution of gray and white matter differ from both the cord and the brain. The pattern is irregular and characteristic of the medulla. All of the spinal tracts pass through the medulla and the cranial nerves from the eighth to the twelfth, except a portion of the eleventh, originate in this structure.

It also contains the various reflex and autonomic centers which control circulation, respiration, the various secretions and the visual movements. The superior and inferior olivary bodies are connected with the cord, the basal ganglia, and the cerebellum. These are concerned with coordination and equilibrium. The pyramidal tracts decussate in the medulla. Disease of the medulla may affect the tracts and nerves passing through it and may cause the various types of bulbar palsy.

The Cranial Nerves

The cranial nerves occur in 12 pairs, they carry sensory, motor or both sensory and motor impulses to various structures and organs, each on its own side of the body Some nerves cross one another and supply opposite sides of the body.

The cranial nerves are:

The first pair, or the olfactory nerves, are concerned with the sense of smell Their fibers run from the olfactory mucous membrane of the nose to the olfactory bulbs in the brain.

The second pair, or the optic nerves, are concerned with sight. They run from the ganglion cells of the retina through the optic chiasm Some fibers of the optic nerves cross in the optic chiasm so that

reflex action in the spinal cord, the medulla or structures of the brain other than the cerebral cortex.

Gnostic sensations reach the cerebral cortex by way of the peripheral nerves through the dorsal roots of the spinal nerves; they ascend the posterior white columns of the cord and ascend uncrossed to the nuclei gracilis and cuneatus in the medulla. The fibers leaving these nuclei cross in the medial fillet (lemniscus) and ascend to the thalamus and from it by way of the posterior limb of the internal capsule and the corona radiata to the somesthetic area of the cerebral cortex in the posterior central gyrus.

Thus a fairly large portion of the cerebral cortex is concerned with perceiving general body sensations This sensory portion lies in the greater part of the *surface of the parietal lobes,* occupying the *postcentral gyrus, the superior parietal lobule* and the part of the *supramarginal and angular gyri*

The gross sensations of pain, temperature and general movements are perceived in the *thalamic region,* but the ability to discriminate between degrees and types of these sensations is the function of the *cerebral cortex. The cerebral cortex* has the ability to identify and discriminate as follows:

(*a*) The degrees of heat, *i. e.,* warm, hot, burning, cool, cold, or freezing.

(*b*) Touch, *i. e,* degree of smoothness or roughness.

(*c*) Identify each of the two sharp points placed closed together upon the surface

(*d*) The direction of small joints, whether displaced upwards, downwards, or laterally.

(*e*) The size, shape, texture, and weight of objects (stereognostic sense).

(*f*) The relations of a stimulus in one, two or three dimensional space.

The special senses such as hearing, sight, smell and taste are conveyed to the brain by special cranial nerves stretching from the special sense organs to definite centers in the brain.

The Cerebellum

The cerebellum, like the cerebrum, has its gray matter externally and its white matter internally It is made up of two *hemispheres, the cerebellar hemispheres,* and a connecting bridge, the vermis The cerebellum contains several motor and sensory tracts that are on their way from the spinal cord to the cerebrum and is also the seat of a number of important functions. It is connected to the brain stem by the inferior, middle and superior peduncles.

Each cerebellar hemisphere receives homolateral impulses from muscles, tendons, ligaments and other deep structures and contralateral impulses through the vestibule. Each cerebellar hemisphere influences the postural activity and muscle movements of its own side of the body. Stimulation of the cerebellum produces flexor attitudes Suppression of cerebellar activity will produce, ipsolaterally, hypotonus, weakness or asthenia of the affected muscles, ataxia, incoordination or asynergy of movement. Disease of the cerebellum or its pathways will produce jerky and misdirected movements such as are seen in chorea, intention tremors, nystagmus, past pointing and pendular patellar reflexes, tremors and postural defects.

Equilibrium and Orientation Pathways: ***The centripetal pathways*** to the cerebellum are Gowers' tract and the direct cerebellar tracts, the pathway from the labyrinth, the inferior olive, and from the cortex.

Lesions and Symptoms of the Pupillary Reflex Arcs

1. Lesions and symptoms of left optic nerve. Pupils are equal; direct light reflex abolished on the side of the lesion and the consensual on the opposite side; illumination of the right retina produces contraction of the left pupil as well as of the right.

2. Lesions and symptoms of the chiasm. Pupils equal; consensual light reflex retained, bitemporal hemianopia; hemiopic pupillary light reflex (?).

3. Lesions and symptoms of left optic tract. Homonymous hemianopia with nasal blindness of the left side; hemiopic pupillary light reflex (?).

4. Lesions and symptoms of left pupillary fibers of the geniculate ganglion. Hemiopic pupillary light reflex without hemianopia (?); bilateral lesion, Argyll-Robertson pupils.

5. Lesions and symptoms of left oculomotor nerve behind the ciliary ganglion; loss of reaction to accommodation on the left side with slight dilatation of that pupil; direct and consensual light reflex in the left pupil abolished.

6. Lesions and symptoms of left ciliary ganglion; same symptoms as at 5.

7. Lesions and symptoms of optic radiations behind the left geniculate ganglion; homonymous hemianopia with nasal field blindness on the left side, Wernicke's hemianopic light reflex, *viz*, light reflex present in both pupils. In bilateral lesions, total blindness with retention of pupillary light reflex on both sides.

8. Lesions of the inhibitory fibers of the medulla, a bilateral section produces a very rapid light reflex, because inhibition is suppressed; should the lesion be irritative, there will be myosis and rigidity to light.

9. Mesial pontine irritative lesion, diminution or suppression of light reflex; destructive lesion, here, as well as at 11, prompt pupillary light reflex reappears and a normal contour of the pupils, as in 8

10. Bulbar hemisection; suppresses inhibition of the contralateral pupil; an irritative lesion produces rigidity to light with myosis in the contralateral pupil.

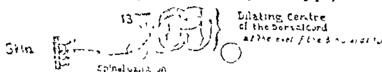
11. Suppression of the sympathetic reaction, myosis.

12. Section of the medulla at this level produces transient dilatation of the pupils; the light reflex is not modified.

13. Lesions and symptoms of communicating ramus of the sympathetic of the first dorsal segment; myosis of the monolateral pupil and no response to cutaneous stimulation of the sympathetic (no dilatation)

14. Section of the cervical sympathetic; same as in 13, the light reflex is not interfered with.

(*According to Bach and Meyer, modified.—Encyclopédie Ophthalmologique*)



Lesions and Symptoms of the Pupillary Reflex Arcs

the fibers of each nasal half of the retina originate in the opposite optic nerve.

The third pair, or the oculomotor nerves, are the great motor nerves of the eyes; each supplies all the muscles of the eyeball except the external rectus and superior oblique.

The fourth pair, or the trochlear or patheticus nerves, supply the upper oblique muscle of each eye (motor).

The fifth pair, or the trigeminus or trifacial nerves, are the great sensory nerves of the head and face Each divides into three main branches: (1) The ophthalmic division; (2) the superior maxillary division; these two are sensory; and (3) the inferior maxillary division which is mixed; that is, both sensory and motor; and a lingual branch. which is concerned with the special sense of taste.

The sixth pair, or the abducens nerves, supply the external recti of the eyes (motor).

The seventh pair, or the facial nerves, are the great motor nerves of the face. Some sensory fibers from the trigemini run with the facials, giving them some sensory function.

The eighth pair, or the auditory nerves, are concerned with hearing and with vestibular function.

The ninth pair, or the glossopharyngeal nerves, contain special fibers for taste, sensation and for motor activity.

The tenth pair, or the pneumogastric or vagus nerves, are mixed sensory and motor. They supply the pharynx and larynx and have numerous connections with the autonomic nervous system, and also with the ninth, eleventh and twelfth cranial nerves and with the first two cervical nerves. They send fibers to the thoracic and abdominal viscera (heart, lungs, kidneys, liver, stomach, intestines, etc.), and also contain vasomotor and secretory fibers.

The eleventh pair, or the spinal accessory nerves, are chiefly motor nerves, though they may contain sensory fibers They join the vagi supplying it with motor and cardioinhibitory fibers, and also send fibers to the trapezius and sternocleidomastoid muscles

The twelfth pair, or the hypoglossal nerves, are the motor nerves of the tongue and also supply fibers to the vagi, linguals, upper three cervical nerves and the sympathetics.

The Spinal Cord (Medulla Spinalis)

The spinal cord is a cylindrical structure composed of nervous tissue and is enveloped by three coats An inner highly vascular delicate coat, the pia mater; a middle coat, the arachnoid, and a fibrous external coat, the dura mater, which extends to the level of the second sacral vertebra ending in a *cul-de-sac* The spinal cord occupies the vertebral column and measures from 40 to 45 cm. in length, extending from the foramen magnum, where it is continuous with the medulla oblongata, to the level of the first or second lumbar vertebra, where it terminates into the conus medullaris. A thin filament extends beyond the conus medullaris, the filum terminale The spinal cord is perforated in the center throughout its length by a central canal.

The cerebrospinal fluid occupies the space between the pia and the arachnoid (the subarachnoid space), and the *cul-de-sac* formed by the dura at its terminal end. The site chosen for a "spinal puncture" is below the fourth or at times below the third lumbar vertebra, which is one to two intervertebral spaces below the termination of the spinal cord and within the *cul-de-sac.*

Lesions and Symptoms of the Pupillary Reflex Arcs

LESIONS AND SYMPTOMS OF THE PUPILLARY REFLEX AREA

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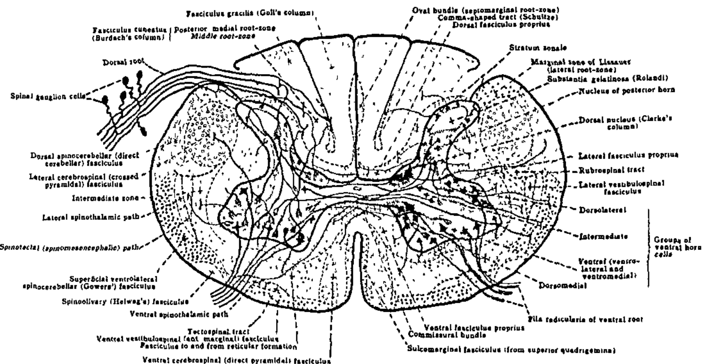


Fig 2—Cross section of spinal cord showing position of the chief descending and ascending tracts (From Morris' Anatomy, P. Blakiston's Son & Co.)

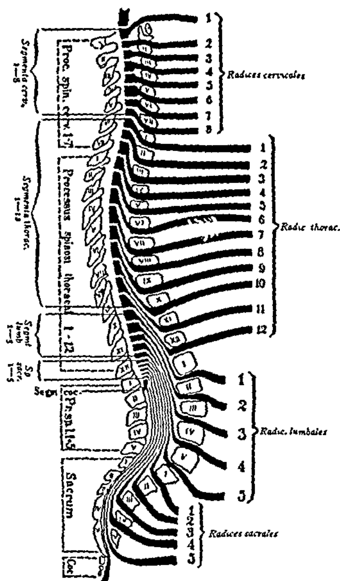


Fig 3—Topographical relations of spinal cord segments to vertebral bodies, spinous processes and points of exit of spinal roots (Reproduced from *Manual of Physical and Clinical Diagnosis* by Otto Seifert and Friedrick Mueller, translated by E. Cowles Andrus, M D, J B Lippincott Co, Philadelphia)

cord and are known as the "anterior or ventral horns" The thin ends of the commas are elongated and are in the posterior part of the cord, they are known as the "posterior or dorsal horns.' The connecting bar is known as the "gray commissure"; it unites both lateral halves of the cord The entire "H" structure and its surrounding white matter run along the entire length of the spinal cord.

The Sensory Tracts of the Spinal Cord: Sensory or afferent impulses, such as touch, pain, and thermal, to-

The external appearance of the spinal cord is whitish in color, and somewhat flattened; on its anterior surface it has a deep median groove, and on its posterior surface a shallow median sulcus which runs the entire length of the cord The cauda equina is made up of the last four lumbar, the five sacral and the coccygeal nerves. Because the spinal cord terminates at the first or second lumbar vertebra, the lower spinal nerves in order to reach their respective intervertebral foramina have to descend vertically in the canal around the conus medullaris and the filum terminale, thus forming the cauda equina

The spinal cord is divided into two lateral halves united to form a more or less cylindrical mass. It has two enlargements, the cervical enlargement, extending from the third cervical to the second dorsal vertebra, and the lumbar enlargement, extending from the ninth thoracic vertebra to the first lumbar.

Spinal Segments: The spinal cord is arbitrarily divided into 31 segments; each segment corresponds to an imaginary line passing through the highest nerve root filaments of each successive spinal nerve.

The spinal cord is also divided according to its relation to the spinal vertebrae Therefore the cervical portion of the cord has 8 segments; the thoracic, 12; the lumbar, 5; the sacral, 5, and the coccygeal, 1.

The Internal Structure of the Spinal Cord: The examination of a cross section of the spinal cord shows it to be composed of two substances, a white substance (substantia alba) occupying the outer portion, and a gray substance (substantia grisea centralis) which is centrally located and is arranged in the shape of an H.

The White Substance: This consists of medullated and some unmedullated nerve fibers imbedded in a spongelike network or neuroglia surrounded by the glial sheath which dips into the cord along with pial septa that carry the cord's blood vessels. The admixture of gray and white matter varies at the different levels of the cord The gray substance predominates in the cervical and lumbar regions while the white matter is most abundant in the thoracic region. While some of the nerve fibers in the white matter run in a more or less transverse direction, such as those crossing from one side of the cord to the other by way of the anterior white commissure, the majority of the fibers run a longitudinal course and are arranged in bundles or tracts and divided into three columns (funiculi) These are: (1) The anterior column lying between the anterior median fissure and the anterior lateral sulcus; (2) the lateral column lying between the anterior and posterior lateral sulci; and (3) the posterior column lying between the posterior median fissure and the posterior lateral sulcus In the cervical and thoracic regions the posterior column is divided by the posterior intermediate sulcus into two parts, a medial one, the fasciculus gracilis or Column of Goll, and a lateral one, the fasciculus cuneatus or Column of Burdach (SEE: Fig 2, p. 817)

The *gray* substance is made up chiefly of nerve cells, dendrites, and unmyelinated as well as some myelinated fibers. It also contains blood vessels and neuroglia. The gray matter is arranged in two comma-shaped masses, one for each lateral half of the cord; both commas are united by a transverse gray bar. The thick ends of the commas are blunt and are in the anterior or ventral part of the

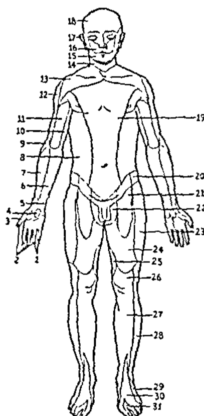


Fig. 4—Radicular distribution of sensory

ris 6, N cutan. antibrachii med 7 N cutan

femor lat. 24, Rami cutan. ant. (n femoralis). 25, Ram cutan. n. obturatorii 26, Ramus infrapatellaris n. sapheni 27, Rami cutan. cruris mediales n saphení 28, N cutan surae lat. (n. peron communis). 29, N cutan surae med (n tibialis) 30, N peron. superfic. 31, N peron profundus

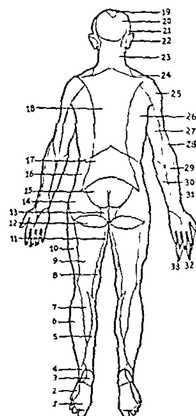


Fig 5—Radicular distribution of sensory areas—posterior 1, N plant med (n tibialis) 2, N plant. lat. (n. tibialis) 3, Rami calcan med. (n tibialis) 4, Rami calcan lat (n. tibialis) 5, Rami cutan. cruris mediales *n.* sapheni. 6, N cutan. surae lat (*n. peron.* comm.) 7, N cutan femor post. 8, Ram. cutan n obturatorii 9, N cutan.

Nn. cutan clun. super (ramor post nn. lumb) 15, Rami post. nn. sacralium 16, Ram. cutan lat. n iliohypogastr 17, Rami post. nn. lumbalium. 18, Rami post nn. thoracalium 19, Trigeminus I. 20, N occipit. *major* 21, N. occipit. minor 22, N auricularis magnus 23, Rami post. nn cervicalium (CIII-VII) 24, Nn. supraclaviculares 25 N cutan. brachii later (n axillaris). 26 Rami cutan. lat. nn intercostalium. 27, N cutan. brachii med. 28, N. cutan. brachii post (n. radialis). 29, N. cutan. antibrachii dors. (n. radialis). 30, N cutan antibrachii med 31, N. cutan. antibrachii lat. (n. musculocutanei). 32, Ram superfic. n radialis. 33, Rami dors. manus n ulnaris

gether with the sensory impulses from the skin, muscles, viscera and joints arise in the peripheral sense organs They are carried by nerves to the spinal cord and enter it by way of the posterior root, thence to be carried along the spinal sensory pathways either to the brain or to a synapse station in the cord. The dorsal or posterior root, as it enters the spinal cord, breaks up into many fibers, *some are medial, others are lateral.* Each fiber divides into two branches, a longer ascending and a shorter descending branch.

The ascending medial branches, which are myelinated fibers, run in the posterior funiculus, some of them reach the medulla and others terminate at various levels in the gray matter of the spinal cord

The descending medial branches, which are also myelinated, are relatively short; some enter the gray matter of the posterior column at once; others descend in *the fasciculus interfascialis or the comma* tract of Schultze; still others reach the posterior median septum and are mingled with descending fibers from cells within the gray matter of the spinal cord.

Collaterals: Fine collateral filaments are given off from the ascending and descending branches; some end in the anterior gray *column and others in the* posterior gray column; still others run through the posterior commissure to the opposite side of the cord, ending in the *posterior column.*

The fibers of the lateral division are unmyelinated; they form the lateral root and enter the tract of Lissauer (dorsolateral fasciculus)

The various sensory pathways in the spinal cord are as follows:

The Dorsal Spinocerebellar Tract (direct cerebellar tract of Flechsig): The fibers arise from the cells of the posterior *nucleus* (*Clarke's column*) and run in the lateral funiculus of the same side and finally reach the cerebellum by way of the inferior peduncle.

Ventral Spinocerebellar Tract (Gower's tract): The fibers arise from the posterior gray column and the intermediate gray matter of the same and opposite side, ascend to the cerebellum by way of the anterior spinocerebellar tracts, and through the superior cerebellar peduncle. "*The path from the periphery to the cerebellum consists of two* neurons with a synaptic *interruption in* the gray matter" (Ranson)

The Lateral Spinothalamic Tract: This consists of fibers originating from cells in the posterior column on the opposite side; they cross the median line in the anterior white commissure and ascend in the anterior funiculus ending in the thalamus. From there the fibers are relayed to the cerebral cortex. They are *believed to be the conductors of pain and* temperature impulses

The Spinoölivary Tract: This arises from the posterior gray column, crosses to the opposite side of the cord, ascends in the ventral funiculus and ends in the inferior olivary nucleus of the medulla

The Spinotectal Tract: The fibers *arise from cells in the gray column of the* cord, cross to the opposite side, ascend in the lateral funiculus with the fibers of the lateral spinothalamic tract and *end in the corpora quadrigemina.*

The Column of Goll (fasciculus gracilis)· This consists of fibers that *originate* from the posterior nerve roots in the lower cord segments; *it lies near* the posterior medium septum, *and increases in size* as it ascends the cord: it terminates in the nucleus gracilis of the medulla oblongata. *It carries* upward

scend from the brain through the medulla and reach the spinal cord uncrossed, so that the fibers in the brain and spinal cord are homolateral or ipsilateral This tract is known as the *direct pyramidal* or the *ventral corticospinal tract.* It is a comparatively small tract; the fibers descend into the spinal cord as direct fibers to a certain level, and then most of them cross in the anterior white commissure so that at their termination they also are crossed fibers. Others terminate on the side of their origin.

In addition to the two pyramidal tracts, we recognize as more or less important the following motor pathways, which constitute the accessory motor or extrapyramidal system

The rubrospinal tract (v. Monakow) arises in the red nucleus; crosses in the decussation of Forel, and descends in the cord near the crossed pyramidal tract

The tectospinal tract originates in the *superior corpus quadrigeminum,* crosses the median line in the decussation of Meynert and descends finally in the anterior column of the cord

The vestibulospinal tract originates in Deiters's nucleus in the bulb, and descends uncrossed in the spinal cord

It is probable that the axis cylinders of most of these tracts end around the anterior horn cells.

The Spinal Nerves

There are 31 pairs of spinal nerves; each pair leaves the spinal cord through its respective intervertebral foramen on either side of the spine, so that each lateral half of the body is supplied by identical nerves.

The spinal nerves are Cervical, 8, thoracic, 12, lumbar, 5; sacral, 5, and coccygeal, 1. The lumbar, sacral and coccygeal nerves form the cauda equina

Nerve Roots: Each nerve is attached to the spinal cord by two roots, a *posterior* or dorsal root, which is *sensory,* and an *anterior* or ventral root, which is *motor.*

The posterior root is the larger of the two. It is attached to the posterolateral furrow of the cord; unites to form two bundles, and contains a spinal ganglion. All sensory impulses from the periphery reach the spinal cord by way of the posterior roots through their ganglia.

The anterior root transmits motor impulses from the cord to the periphery; it leaves the spinal cord by way of its anterior surface in a number of filaments which unite to form two bundles near the intervertebral foramen

Each of the cerebral and spinal nerves is made up of lesser nerves which supply the various structures of the body with sensory and motor sensitivity The largest spinal nerves and nerve roots are attached to the cervical and lumbar portions of the spinal cord; these supply the upper and lower extremities respectively.

Spinal and Peripheral Localization

Every muscle of the extremities is innervated by fibers emanating from two or more spinal roots

Every area of sensory cutaneous distribution is supplied by three spinal roots, one root being principal and predominating

Peripheral nerve distribution is different from segmental nerve distribution Hysterical anesthesia does not correspond to either of these distributions and, in addition, often tends to assume a stockinglike or glovelike form when it involves the extremities

The following rule, formulated by Ziehen, is useful in the determination of

sensory impulses from the lower extremities and the lower half of the body

The Column of Burdach (fasciculus cuneatus). The fibers constituting this tract also originate from the posterior nerve root fibers, but at a higher level, that is, from the thoracic and cervical regions Some of the fibers ascend but a short distance and end in the gray matter; others ascend to the medulla oblongata and terminate in the cuneate and gracile nuclei. It carries sensory impulses upward from the upper half of the body and upper extremities Fibers from both the gracile and cuneate nuclei decussate in the medial lemniscus and proceed to the thalamus and thence to the cerebral cortex.

Motor, Efferent or Descending Tracts of the Spinal Cord: The motor pathways have their origin in various parts of the brain and the fibers from the motor neurons descend into the spinal cord, forming the motor tracts The impulses thus originating in the various parts of the brain are transmitted downward to the spinal cord, and are further carried to their destination by way of the anterior roots of the spinal nerves by the peripheral nerves

The Pyramidal Tracts: The principal motor pathways are the pyramidal tracts, the crossed and the direct pyramidal tracts. The fibers of these tracts arise from the large pyramidal cells (Betz cells) of the motor regions of the cerebral cortex (precentral gyrus); they pass down as direct fibers, one on each side, through the subjacent levels of the brain. As they reach the lower level of the medulla (the decussation of the pyramids), some of the fibers cross (decussate) from one side to the other, so that when they reach the spinal cord the fibers from the left side of the brain are in the right side of the cord and those from the right side of the brain are in the left side of the cord. The crossed fibers form the

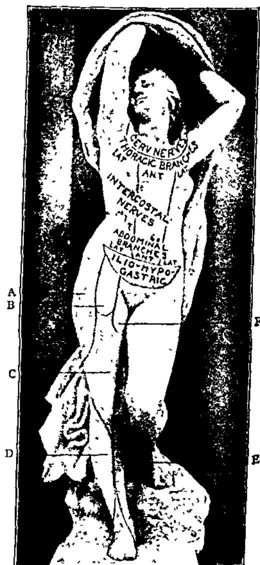


Fig. 6—Showing the distribution of the sensory nerves of the skin, anterior aspect of trunk and leg A, External cutaneous. B, Genito-crural C, Anterior crural. D, External popliteal. E, Long saphenous F, Ilio-inguinal (From Butler.)

crossed pyramidal or the *lateral corticospinal tract.*

Other fibers originating from the pyramidal cells in the motor area de-

loss of all these sensations in the area supplied by the affected nerve.

Spinal Cord Lesion: The various sensory impulses that reach the spinal cord travel in special or individual pathways. Thus touch and pressure travel upward by many paths within the spinal cord; painful impulses travel upward by another path, the spinothalamic tract; and impulses from muscles, joints and tendons travel upward by still another path, the posterior columns. Therefore, a localized spinal lesion may affect only some of the sensations in the area supplied by the cord segment It, however, shows great selectivity of involvement with motor disturbances and frequently also cerebellar symptoms.

Vegetative or Involuntary Nervous System

Sympathetic and Parasympathetic

The vegetative nervous system is composed of two divisions, the sympathetic and parasympathetic In their origin, to some extent in their anatomic structure and in their functions, they appear to be in opposition to each other

The sympathetic division of the vegetative nervous system causes dilatation of the pupil, dryness of the skin, rapid heart action, dilatation of the sphincters, dilatation of the pial vessels, slowing of peristalsis, and low gastric acidity

The parasympathetic division causes contraction of the pupil, sweating, slowing of the heart, contraction of the vessels of the pia, contraction of the sphincters, hyperperistalsis and gastric hyperacidity They also differ in their reaction to certain drugs and hormones.

The sympathetic division of the vegetative nervous system (the dorso-lumbar autonomic system) consists in part of the lateral chains of sympathetic ganglia and their connecting fibers. The ganglia are connected with the spinal nerves by the white and the gray rami communicantes. In addition, the sympathetic system includes the three cervical sympathetic ganglia, and the lumbar and sacral ganglia, together with the peripheral plexuses formed by the fibers proceeding peripherally from these ganglia The fibers pass as preganglionic fibers to the cells of the lateral ganglia, where they are interrupted, lose their myelin sheath, and pass as postganglionic fibers to the periphery.

The Parasympathetic or Autonomic Division of the Nervous System (the cranio-sacral autonomic system) This consists of midbrain, bulbar and sacral nerve fibers which supply the same organs and tissues as does the sympathetic system, but whose action is opposite to that of the sympathetic system

The parasympathetic system is divided into three parts· The mesencephalic, the bulbar and the sacral

The mesencephalic corresponds to the oculomotor nerve and nuclei. Fibers from its center and from cervical sympathetic end in the ciliary ganglion,

Bulbar fibers run to some degree with *the facial (fibers to the submaxillary gland)* and glossopharyngeal nerves (fibers to the parotid gland), and to a greater degree with the *vagus nerves* which supply nearly all of the thoracic and most of the abdominal viscera.

Sacral fibers run in the internal pudendal nerve and to the organs supplied by it, *i e*, the lower part of the intestine, the bladder and the genitals.

Sympathetic Influence on Voluntary Muscles: Recent views regard the

the levels of origin of the cervical and thoracic nerve roots from the spinal cord. For the cervical nerves subtract one from the number of the nerves, and

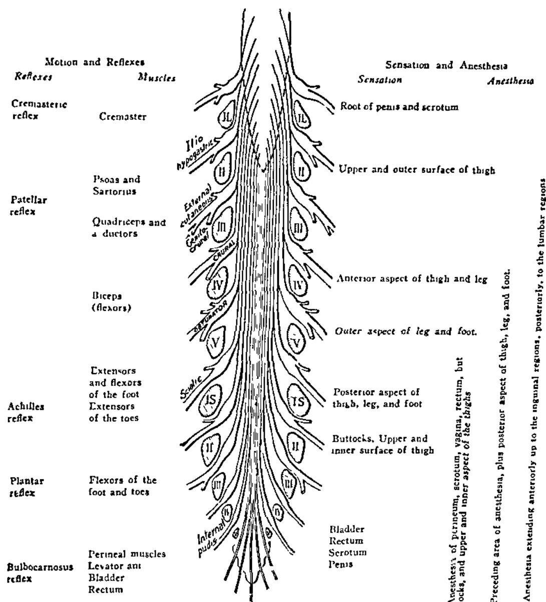
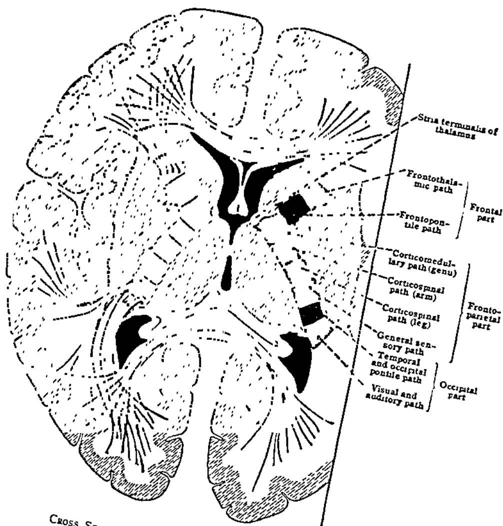


Fig 7—Spinal localization

the remainder will indicate the corresponding spinous process; for the first to the fifth thoracic nerves subtract two; for the sixth to the twelfth thoracic nerves subtract three.

Differentiation Between a Spinal Nerve Lesion and a Spinal Cord Lesion: ***Spinal Nerve Lesion:*** Because a spinal nerve contains all types of sensory fibers through which are transmitted sensations of heat, cold, touch, pain, pressure as well as muscle, joint and tendon sensibility, a destructive lesion in a spinal nerve will cause



CROSS SECTION OF CEREBRUM SHOWING INTERNAL CAPSULE
(From Morris)

voluntary muscles in general as having sympathetic as well as ordinary spinal and cranial nerve innervention.

Sensory fibers from the viscera run through the sympathetic ganglia to the posterior roots of the spinal nerves, where they enter posterior root cells, the with the sympathetic system. The white ramus communicans is interrupted in a spinal ganglion, the fibers lose their myelin sheath and the new unmyelinated fibers reënter the spinal nerve through the gray ramus communicans to supply the arterial system.

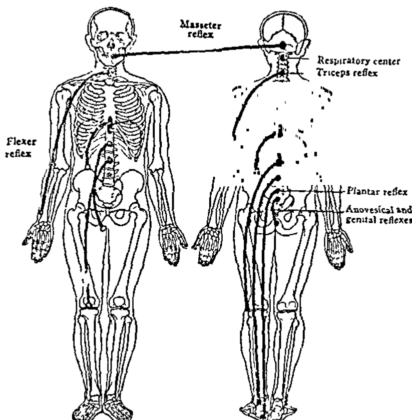


Fig 8—Reflex centers

central processes of which enter the spinal cord. They come into association in the ganglia with cells whose peripheral processes supply skin areas which are often distinctive for the lesion of the viscus in question. Pain due to disturbance of this viscus is often referred to the periphery, the skin area thus becoming the apparent seat of the pain.

The vasomotor centers, especially the vasoconstrictor centers, are associated

Nomenclature: Langley, who has contributed largely to the subject of the autonomic nervous system, adopted a terminology somewhat different from that used here (Gray's anatomy), and still different from that used by Meyer and Gottlieb This has led to considerable confusion, as shown by the arrangement of the terms in the following columns. Gaskell has used the term Involuntary Nervous System.

Involuntary Nervous System

Gray[1]	Langley	Meyer and Gottlieb
Sympathetic nervous system.	Autonomic nervous system	Vegetative nervous system
Craniosacral sympathetics	Parasympathetics.	Autonomic.
Oculomotor sympathetics	Tectal autonomics.	Cranial autonomics.
Facial sympathetics. Glossopharyngeal sympathetics. Vagal sympathetics.	Bulbar autonomics.	
Sacral sympathetics.	Sacral autonomics.	Sacral autonomics
Thoracolumbar sympathetics.	Sympathetic thoracic autonomic.	Sympathetic.
Enteric.	Enteric.	Enteric.

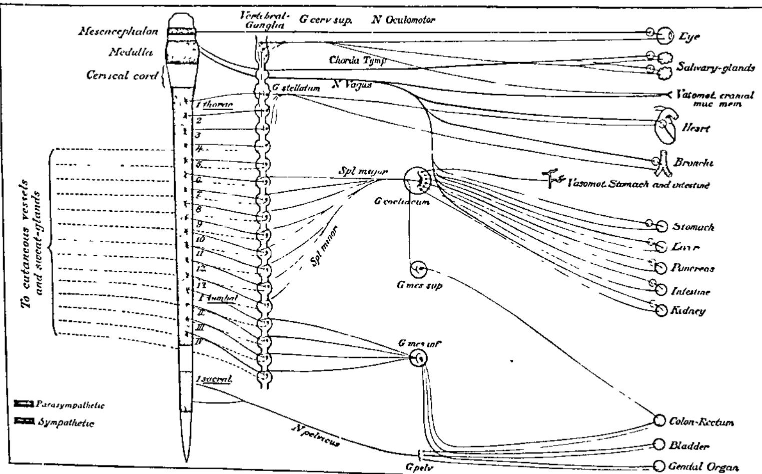
Eppinger and Hess, applying the physiological facts of Langley to clinical medicine, have elaborated upon the theory of autonomic ataxia advocated by Solomon Solis-Cohen in 1892, namely, that the vegetative system is divided into two parts: (1) The autonomic, corresponding with the parasympathetic, or the cranial and sacroautonomic of Langley's classification; and (2) the sympathetic, or the thoracicolumbar portion of Langley's autonomic system. Eppinger and Hess believe that the parasympathetic and sympathetic systems are controlled by the endocrine glands and that normally a balance exists between the parasympathetic and the sympathetic systems, and that this balance may be disturbed so that one or the other of the systems predominates. This would give rise to two opposing conditions: (1) Vagotonia and (2) Sympathicotonia.

1. *Vagotonia* is characterized by nervousness, pale, greasy skin, often spotted with red blotches; sweating occurs easily; hypersalivation; the pupils are small; sinus irregularity and slow pulse rate are often present The bowels are usually constipated; indigestion, hypersecretion and pyloric spasm may occur. Adults may suffer from asthma and eosinophilia. Children may suffer from enuresis and laryngismus stridulus, and from hypertrophy of the vessels and the lymphoid tissue.

2. *Sympathicotonia* presents a picture the reverse of the above; the pupils are dilated; the pulse is rapid; the cutaneous vessels are contracted; the erector pili muscles and sweat glands are hypersensitive. The general response to pain is greatly lessened The sympathicotonic is usually made worse by the injection of epinephrine, while the vagotonic is often relieved by the injection of epinephrine and made worse by the administration of pilocarpine and physostigmine.

Action of Some Drugs in the Sympathetic and Parasympathetic Systems: *Epinephrine* acts as a stimulant on the sympathetic system (except on the

[1] Gray's Anatomy. 1930, p 966, 22nd Edit., edited by Lea & Febiger, Philadelphia and New York.



SCHEME OF THE VEGETATIVE NERVOUS SYSTEM
(Reproduced from Falta's "Endocrine Diseases," edited by Myers,
after Myers and Gottlieb's "Die experimentelle Pharmakologie, etc" The autonomous innervation is colored blue, the sympathetic red)

Involuntary Nervous System

Gray[1]	Langley	Meyer and Gottlieb
Sympathetic nervous system.	Autonomic nervous system	Vegetative nervous system
Craniosacral sympathetics.	Parasympathetics	Autonomic.
Oculomotor sympathetics	Tectal autonomics.	Cranial autonomics.
Facial sympathetics. Glossopharyngeal sympathetics. Vagal sympathetics	Bulbar autonomics.	
Sacral sympathetics.	Sacral autonomics.	Sacral autonomics
Thoracolumbar sympathetics.	Sympathetic thoracic autonomic	Sympathetic.
Enteric.	Enteric.	Enteric.

Eppinger and Hess, applying the physiological facts of Langley to clinical medicine, have elaborated upon the theory of autonomic ataxia advocated by Solomon Solis-Cohen in 1892, namely, that the vegetative system is divided into two parts: (1) The autonomic, corresponding with the parasympathetic, or the cranial and sacroautonomic of Langley's classification; and (2) the sympathetic, or the thoracicolumbar portion of Langley's autonomic system. Eppinger and Hess believe that the parasympathetic and sympathetic systems are controlled by the endocrine glands and that normally a balance exists between the parasympathetic and the sympathetic systems, and that this balance may be disturbed so that one or the other of the systems predominates. This would give rise to two opposing conditions: (1) Vagotonia and (2) Sympathicotonia.

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[1] Gray's Anatomy, 1930, p 966, 22nd Edit, edited by Lea & Febiger, Philadelphia and New York.

sweat glands) and on organs on which the sympathetic has a stimulant action. It does not act on the organs on which the sympathetic system has an inhibitory action, nor does it act on the autonomic system.

Ergotoxin has an effect on the sympathetic system generally the opposite of that of epinephrine

Atropine has a paralyzing effect on the nerve terminations of the parasympathetic system.

Acetylcholine stimulates the parasympathetic system

Pilocarpine has a stimulating effect on the nerve terminations of the parasympathetic system It also stimulates the secretion of the sweat glands.

Neurologic Examination

History

It is as important to obtain a comprehensive history from the sufferer of nervous derangements as it is from patients suffering from other ailments. The history may be elicited directly from the patient or at times, since misleading statements may be made by the nervous patient because of lack of comprehension, willful misrepresentation or spiteful taciturnity, it is preferable to obtain the information from a relative or attendant in the absence of the patient.

It is important when examining young patients to investigate the *previous history* as to manner of birth, instrumental or otherwise, as to lactation, dentition, previous diseases, habits and inclinations as to playfulness, moroseness, precocity, hobbies, fears, anxieties, behavior toward his playmates, sexual impulses, as well as to his schooling, progress at school, studiousness, etc

Family history as to consanguinity, the mental and physical state of near relatives, etc, should be obtained.

The *present complaint* as to onset and general cause are best recorded in the patient's own words, and all symptoms described by him are to be examined in detail Inquiry is also to be made as to headache, digestion, vomiting, convulsions, sleep, dreams, disorders of sensation and of special senses.

Physical Examination

Having elicited a thorough history, the physical examination is then carefully made. The physical examination consists of general examination, local examination, and various special examinations.

The General Examination

This commences just as soon as the patient enters the examiner's presence. In ambulatory patients, the general appearance, build, nutrition, color, behavior, manner of dress, gait, posture and the general intelligence should be noted In bed patients, posture, restlessness, mentality and general behavior are important observations.

The Local Examination

Head: This includes examination of the *skull* as to size, shape, and evidence of deformity or of injury. The *head* is examined for the amount of hair, its color and texture, abnormal pulsations, tumors, depressions and rashes. The *face* is examined for expression, mobility, scars and edema; the *eyes* for the size of the palpebral fissures (wide, narrow, equal), for ptosis, tremor of the eyelids, and lagging during ocular movement. The

eyeballs are examined to determine whether they are prominent, protruding, or sunken, and for the presence of strabismus, mobility, static nystagmus, difference in the colors of the irides, the dimensions and form of the pupils, pupillary inequality, also for the reaction of the pupils to light, accommodation, and convergence Notice is to be taken of the symmetry of the frontal wrinkles and of the nasolabial folds, the thickness of the lips, tremor and retraction of the lips, immobility of the facial muscles in repose, and of fibrillary contractions or spasms.

The Mouth: The following should be observed: Pharynx, dentition, size of tongue, its position in the mouth and manner of protrusion, position of the uvula, movements of the velum on phonation and on irritation

Neck: The position of the head, the presence of rigidity of the neck, the presence of enlarged glands, scars, or lesions, and the presence of any tics or spasms should be noted

Examination of the thyroid gland is important

Shoulder Girdle, Chest and Upper Extremities: The *shoulder girdle*, the *upper extremities*, and the *chest* are examined for size, shape, and symmetry, and the condition of the muscles; the *hands* are examined for their size, shape, sensitivity, strength, musculature, reflexes and for the presence of contractures

Pelvic Girdle and Lower Extremities: The *pelvic girdle* and *lower extremities* are examined as to the position of the limbs in the dorsal position of the patient, length of the limbs, contractures, condition of the muscles, size of the feet and their form, and the presence of any deformities or contractures

Notice is to be taken of the position of the lower limbs when the patient stands erect, the static position of the pelvis, the increase or diminution of the lumbar lordosis, and the symmetry of the folds of the buttocks.

Trunk: The *trunk* is examined for size, shape, posture and nutrition; and for kyphosis, scoliosis and lordosis of the spine.

Skin: The following should be noted: Subcutaneous tissue, nails, color of the skin, its thickness, temperature, moisture, venous network, pigmentation, edema, ulceration, general or local increase of the fat tissue, tumors, exanthemata, acrocyanosis, and the presence of malformations of the nails.

Special Examination

Reflexes: Percussion of the tendons and of the bones is carried on for the provocation of the tendon and periosteal reflexes Tickling is employed to provoke the mucous membrane reflexes and light stroking to elicit the skin reflexes, which, however, are readily exhausted (SEE p 831).

The reaction of the pupillary reflexes to light (homolateral and contralateral reflexes), to convergence, to accommodation, and to pain is to be tested

Sensibility: The eyes of the patient should be closed The sensibility is examined by the use of Weber's compasses, the examiner's finger, and by tests for the localization of touch.

Sensibility to Pressure: This is examined either roughly, by judging the amount of pressure applied, or by the use of a baresthesiometer. Can the patient detect light touches, such as cotton?

Thermic Sensibility: This is tested by the use of large test tubes containing hot and cold water, or by a hot and

a cold spoon, or by any other hot and cold object.

Pain Sensibility: When the point or the head of a pin is applied to various parts of the body, the patient is asked to distinguish between the point and the head. An algesimeter may be substituted for a pin.

Skin Sensibility to Electricity: The faradic current is used, tingling being the normal sensation to light currents. As the strength of the current is increased, a painful sensation appears. A large electrode is used for the back and a small electrode for the part to be tested.

Muscle-joint Sense: Various muscles and joints are moved passively by the examiner.

Bone Sense: A tuning fork of 128 vibrations a second is applied to the bone surface, and the sense of vibration noted by the patient.

Stereognostic Sense: Is the object in the hand recognized by name? If not, can its attributes to touch be described? The stereognostic sense is not simple, inherited, and primary, but complex, acquired, and secondary

Palpation and Percussion of the Nerve Trunks and of the Muscles: The nerves should be palpated for pain in their entirety and at their point of exit from muscles and bony canals (points of Valleix). It should be noted, too, whether the nerve trunks are painless where compression ought to cause a certain degree of pain, as, for example, in tabes.

Muscles should be palpated to determine their size, consistency, and whether they are tender.

Nerve trunks should be tapped with the percussion hammer to ascertain whether there is any response. In tetany there is hyperexcitability of the nerve trunks to mechanical stimuli.

Muscles respond to tapping in two ways: (*a*) With contraction *en masse* dependent upon the integrity of the nerves supplying the muscle. (*b*) With local contraction at the point of percussion (forming momentarily a ridge—idiomuscular contraction), dependent on the excitability of the muscle fibers themselves and independent of the control of the nerves The mechanical excitability of the muscle is increased in tetany, in certain neuritides and in chronic wasting diseases, such as tuberculosis It is decreased in muscular dystrophy.

Examination of Motility: ***Active Motion:*** *Face.* The closing and opening of the eyelids, movements of the eyeballs (lateral movements, up and down movements, circumduction, convergence), wrinkling of the forehead, various movements of the facial muscles (if possible, with and without emotional expression) are to be observed.

Mouth, Pharynx, Larynx: The opening and closing of the mouth, movements of the jaw, testing of the force of the muscles of mastication, protrusion of the tongue, movements of the tongue, movements of the palate during phonation, movements of the pharyngeal wall during phonation, deglutition of fluids and of solids should each be noted. Laryngoscopy is valuable.

Movements of the Head and Upper Extremities: The movements of the head, the shoulder girdle and the upper extremities should each be executed separately.

Movements of the Trunk: The respiratory and abdominal movements, the method of rising from a supine to a seated posture, the pelvic girdle and the

movement of the lower extremities should be observed.

Gait: The posture of the trunk during walking, the method of planting the feet, the direction of walking when an attempt is made to walk along a straight line, and the kind of reversal of direction at command that the patient can make are to be noted (SEE: pp 120 and 851).

Passive Motion: Passive movements of parts should be carried out when the patient relaxes his muscles, and hypertonia, contracture, and hypotonia noted

Examination of Coördination: ***Dynamic Coordination:*** The execution of movements that require precision, at first with the eyes open, then with the eyes closed, are to be compared

Static Coördination: The erect station on both feet, close together, then on one foot, with the eyes open and then with them closed, is to be observed The patient is to be asked to raise his lower extremities while he lies supine and to raise his upper extremities to form a right angle with the trunk The examiner should note how long the patient can maintain these positions. The time is decreased in cerebellar lesions

Examination of Orientation and Equilibrium: These require paraphernalia. A rough test is the ordinary one of past pointing; the patient endeavors to touch with one finger, when his eyes are closed, his nose, the corresponding finger of the other hand, or the extended finger of the examiner

Electrical Examination: In examining the electrical reaction of the nerves and muscles one needs an induction apparatus and a galvanic battery capable of yielding a current strength of at least 30 milliamperes, some means of interrupting the current, preferably by a break contrivance attached to one of the electrodes, a pair of cords, a large and flat electrode (60 sq. cm.), which is applied to the sternum or the back, and a small electrode for application to the point to be tested (SEE: *Electrical Reaction*, p. 848).

Examination of the Genitourinary System: The functions of the sphincters of the bladder and of the rectum should be investigated and tests made of these, perhaps by specialists. The sexual life of the individual should be tactfully probed, at least as much as is necessary to explain the symptoms The question of psychoanalysis is a mooted one, and need not be entered into here (SEE: *Symptoms of Mental Diseases*, p 885).

Miscellaneous Examinations: Many organic diseases are associated with mental symptoms, and the practitioner should have at least an elementary knowledge of psychiatry in order that he may be able to detect some of them and to recognize their importance if not their significance

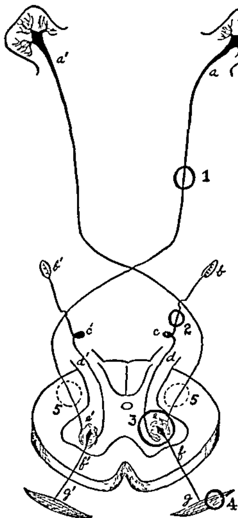
Laboratory methods, such as examination of the cerebrospinal fluid, are valuable aids to neurologic diagnosis.

Of late, ventriculography, encephalography and electroencephalography have become important aids in the localization of the brain and spinal cord lesions *Queckenstedt's sign*, or the absence of an increase in cerebrospinal fluid pressure upon compression of the cervical vessels or the abdominal aorta, usually signifies a spinal block, and may be indicative of a tumor of the cord.

Reflexes

A peripheral stimulation that results in a muscular contraction or in glandular activity is known as a reflex. Reflexes may be divided into the tendon, the osteoperiosteal, the cutaneous, and the

mucous. The first two of these are produced by percussion of a tendon or of a bone, the last two by stimulation of a cutaneous region or of a mucous membrane. Certain other reflexes are spoken of as visceral



anterior horns, second part of the reflex

muscle *response to all stimuli, and flaccidity*; at 5, same as at 1.

A simple reflex arc consists of a peripheral end organ and its afferent fiber and cell, an efferent fiber from this cell, an intermediary cell with its efferent fiber and the muscle (with the muscle end plate). The spinal cord is the seat of this simpler reflex fiber activity. In a sense all nervous activity that involves the transmission of an impulse from one neuron to another is reflex. In other reflexes lower cerebral centers are involved. *The spinal centers are subject to the inhibitory action from the cerebral and cerebellar centers, especially when the impulses are conveyed by the pyramidal tract*

After a transverse section of the spinal cord, reflex activity is abolished for a time (period of shock), recovery takes place, and is followed by a period of increased reflex activity below the level of section.

The destruction of a portion of a reflex arc is followed by complete loss of the tendon and skin reflexes. This occurs: (*a*) *In peripheral neuritis*; (*b*) in tabes dorsalis; (*c*) in anterior poliomyelitis and all the acute and chronic destructive processes involving the anterior horn cells; (*d*) in cases involving destruction of the posterior horn cells (syringomyelia, hematomyelia).

Irritation of a portion of the reflex arc produces increased reflexes. This occurs in certain forms of neuritis and of radiculitis, in strychnine poisoning, and in tetanus.

Total transverse section of the spinal cord at various levels produces abolition of reflexes presided over by that section of the cord.

Alterations in the pyramidal tract produce increase of the tendon reflexes. This is seen in:

(*a*) Meningoencephalitis, cerebral tumors, cerebral compression, and in cerebral thrombosis, embolism, and hemorrhage; in the latter three usually after the initial period of shock, although spastic phenomena may occur early.

(*b*) Spinal compression where a condition resembling that of spinal section may be obtained

(*c*) Degenerative diseases of the cord, primary (amyotrophic lateral sclerosis, primary lateral sclerosis), or secondary (myelitis, meningomyelitis, spinal arteriosclerosis).

(*d*) Disseminated sclerosis

In affections of the central neuron there is an antagonism between the tendon and the cutaneous reflexes While the tendon reflexes are increased, the skin reflexes are often diminished or abolished.

Pflueger's Law: 1. The reflex occurs upon the same side of the body to which the irritant is applied, and in muscles the motor nerves of which rise from the same segment of the cord.

2. If the reflex occurs on the opposite side, only the corresponding muscles contract.

3 If the reflexes are unequal on the two sides, the stronger reflexes are on the side to which the irritant has been applied

4 When the reflexes extend to the other segments, the direction of the extension is toward the medulla

5. All the muscles of the body may yield reflexes

The reflex arc may be broken in any one of the following ways (*a*) When the sensory nerve does not conduct the impulse toward the center; (*b*) when the sensory cell is impaired so that it cannot receive the impulse, (*c*) when the motor cell is impaired so that it cannot receive the impulse, (*d*) when the motor nerve is impaired so that it cannot transmit the motor impulse.

In most, if not all, reflexes intermediary neurons are also involved. A reflex may become exaggerated when the motor cells from which fibers supplying the parts in question are irritated.

Reflex acts are *inhibited* and *modified* by inhibitory impulses passing down from the brain along the inhibitory nerve fibers of the pyramidal tract, and are *increased* or *exaggerated*, or *quickened* when this inhibitory action is removed or reduced by destructive disease that involves the pyramidal tracts.

The spinal centers for the reflexes (variously stated by authors) are as follows

Biceps Fifth and sixth cervical segments

Radial Fifth and sixth cervical segments

Triceps Sixth and seventh cervical segments

Ulnar· Seventh and eighth cervical segments.

Knee Second, third and fourth lumbar segments.

Achilles· Fifth lumbar, first sacral segments

Adductor: Second, third and fourth lumbar segments.

Semitendinosus and semimembranosus: Fourth and fifth lumbar, first sacral segments

Cremasteric· First and second lumbar segments

Scapular. Fifth cervical to first dorsal segments

Cuboid: Fourth and fifth lumbar, first sacral segments.

Epigastric: Seventh, eighth and ninth dorsal segments.

Mesogastric: Ninth and tenth dorsal segments.

Hypogastric: Twelfth dorsal segment.

Plantar: First and second sacral segments.

Gluteal: Fourth and fifth lumbar, first sacral segments.

Anal: Fifth sacral segment

Classification of Reflexes: We usually speak clinically of three groups of reflexes. (I) Cutaneous or superficial reflexes; (II) tendon or deep reflexes; (III) visceral reflexes. Occasionally the vasomotor and osteoperiosteal reflexes are classified separately as are also the cranial reflexes

Cutaneous (and Mucous) or Superficial Reflexes: ***Palatal:*** When the mucous membrane of the palate or when the fauces is touched, the palate draws up. This reflex is *lost* in *bulbar paralysis, postdiphtheritic paralysis,* and *tumors* of the *cerebellopontine angle* When the patient is requested to say *AH*, the palate remains motionless, either unilaterally or bilaterally.

Scapular: When the interscapular region is irritated, the scapular muscles contract This reflex depends upon the integrity of the fifth cervical to first thoracic segments

Epigastric: When the skin of one side of the chest below the nipple is gently stroked, the epigastrium upon that side will retract This reflex depends upon the arcs of the seventh to the ninth dorsal segments.

Abdominal: When the costal margins are stroked downward in the midclavicular line, the abdominal muscles on the same side contract. This depends upon the arcs from the ninth to the twelfth dorsal segments. They are typically lost in pyramidal tract affections and in multiple sclerosis.

Cremasteric: The testicle on the same side draws upward. This depends upon the first and second lumbar segments.

Gluteal: When the skin of the buttock is stroked, a contraction of the gluteal muscles on the same side follows It is controlled by the fourth and fifth lumbar and first sacral segments

Plantar: When the sole of the foot is irritated or tickled, the toes bend plan-



Fig. 10—Technic for eliciting Babinski's reflex.

tarly. This reflex depends upon the integrity of the lower end of the cord (*conus medullaris*) It may be absent normally or after taking of sedative drugs, such as the bromides.

Brissaud's Reflex (or reflex of the tensor of the fascia lata) · This is associated with the plantar reflex and is shown by a contraction of the fibers of the fascia lata in the external regions of the thigh when the sole is stroked.

Pupillary Skin Reflexes: When the chin or neck is stroked, dilation of the pupils follows

Babinski's: When the sole of the foot is stroked upward and inward from

the outer margin, extension of the great toe and a tendency to fanning and spreading out of the other toes are noted This is due *to disease of the pyramidal tract,* seen in hemiplegia and spastic paraplegia due to any cause and occa-



Fig 11—Technic for eliciting the Gordon reflex

sionally in fracture of the skull, uremia and general paresis It is a pathologic reflex, except in infants

Gordon's: When deep pressure is made through the calf muscle on the deep flexor muscles, dorsal flexion of the great toe occurs. Like the Babinski reflex and Oppenheim's, it denotes pathology. It cannot be considered a skin reflex but is mentioned here for convenience

Oppenheim's: When the portion of the tibia just behind the posterointernal border is stroked from above downward, dorsal flexion of the toes occurs. It should hardly be classed with the skin reflexes. It is seen in lesions of the pyramidal tract.

These last three reflexes are abnormal, the most reliable one is the Babinski. Its presence indicates disease of the central motor neurons. This reflex, however, is often noted in normal infants

Sign of Adduction of the Foot (Marie and Meige): Irritation of the internal part of the sole produces contraction of the tibialis anticus muscle and adduction of the foot. This is sometimes found in cortical conditions associated with exaggeration of the tendon reflexes.

Anal: When the anus is irritated with a pin, contraction of the sphincter ani results.

Umbilical: When the side of the abdomen is irritated, the umbilicus moves toward that side This is really a unilateral abdominal reflex

Corneal or Conjunctival: When the cornea or conjunctiva is irritated, closing of the eyelids results

Nasal: When the mucous membrane of the nose is irritated, sneezing will result.

Pharyngeal: When the pharynx is irritated, retching or gagging will result

Paralytic Hyperemic Reflex (dermographia) · When a hard object is drawn over the skin, it will cause congestion followed by ischemia (local anemia). This is really a vasomotor reflex.

Pilomotor Reflex: Erection of the hair follicles takes place when the skin is stroked or exposed to cold (chill).

Defense Reflexes: See under *Tendon Reflexes*

See also under *Vasomotor Reflexes* below The strict vasomotor reflexes are

not concerned with activity of voluntary muscles, as are most of the clinical skin reflexes

Tendon or Deep Reflexes: ***Knee jerk:*** A sudden extension of the knee will occur when the ligamentum patellae is sharply struck, while the leg is crossed over its fellow



Fig 12—Knee jerk with ulnar surface of hand

The knee-jerk reflex is *increased* in: (*a*) Organic disease of the brain; (*b*) incomplete transverse lesion of the cord, above the lumbar enlargement; (*c*) disseminated cerebrospinal sclerosis, lateral sclerosis, sclerosis that is predominantly lateral, earlier stages of combined sclerosis; (*d*) also in mania, hysteria, strychnine poisoning, tetanus, meningitis, and in persons who are "high strung" or fatigued.

The knee jerk is *diminished* or *absent* in: (*a*) Degeneration of the muscle; (*b*) pseudomuscular hypertrophy; (*c*) neuritis, which cuts off the impulse from the cord; (*d*) locomotor ataxia, or any other lesion of the posterior column of the cord; (*e*) poliomyelitis, (*f*) advanced myelitis; (*g*) lesions of the cauda equina or of the lumbar enlargement; (*h*) muscular dystrophy involving the crureus muscle; (*i*) Friedreich's ataxia and combined sclerosis (except in the early stages when it is increased), (*j*) poisoning from certain drugs, *i. e.*, antimony, chloral, or opium; (*k*) pernicious anemia, and (*l*) occasionally it occurs idiopathically.



Fig. 13—Technic for eliciting the patellar reflex with rubber-tipped hammer.

Ankle Clonus: Oscillation of the foot takes place when it is suddenly flexed.

This reflex is elicited in the following manner: The patient is seated, the examiner supporting with one hand the tendo Achillis, while with the other hand he strongly flexes the foot upward, exerting pressure upon the front part of the sole This reflex is often



Fig 14—Technic for eliciting the *Achilles' reflex*

found in lateral sclerosis or spastic paraplegia, in lesions of the pyramidal tract, and in reflex hyperactivity The reflex center is in the fifth lumbar and first sacral segments. It may be absent even when Babinski plantar reflex is present.

Tendo Achillis Reflex (normal reaction). Sudden plantar flexion of the foot occurs when the tendo Achillis is sharply struck. This reaction is increased in lesions of the central motor neurons which cut off the inhibitory action of the brain; also in lesions of the pyramidal tract Its center is the fifth lumbar and first sacral segments. Its absence is an *important early sign of tabes dorsalis* It is absent in pelvic tumors, multiple *neuritis (diabetes, gout, alcohol, metallic poisoning)*, diabetic pseudotabes, and tabes dorsalis

Kernig's Sign: This is resistance to sudden extension of the knee. This reflex is best obtained in the following way:

The patient lies on his back, the leg flexed upon the thigh and the thigh flexed upon the abdomen. The leg is then grasped by the examiner at the tendo Achillis and an attempt made to raise it. When the leg is brought at right angles to the thigh or thereabouts, resistance will be encountered. The presence of this reflex usually indicates meningitis Contraction of the hamstring muscles may also be due to sciatica, and hip- or knee-joint diseases

Dorsal Foot Reflex (Mendel-Bechterew reflex) · Sudden extension of the toes when the dorsum of the foot is



Fig 15—Technic for eliciting Kernig's sign

struck over the fourth and fifth metatarsal bones is usually due to a lesion of the pyramidal tract. Its reflex arc is in the fifth lumbar and first sacral segments.

Biceps Reflex: A contraction of the arm is obtained by striking the biceps *tendon at the elbow*. The patient's fore-

arm rests upon the examiner's arm, palm upward, the elbow joint is supported by the examiner's hand so that the thumb rests in the cubital fossa. With a pleximeter hammer in the free hand, the examiner taps his own thumb smartly. The reflex arc is in the fifth and sixth cervical segments.



Fig 16—Technic for eliciting the biceps reflex.

Triceps Reflex: This is the extension of the arm when the triceps tendon is struck above the olecranon. The elbow is supported by the examiner so that it rests easily with the olecranon upward, while the triceps tendon is struck directly with the pleximeter hammer. The reflex arc passes through the sixth and seventh cervical center.

Maxillary Reflex: This is the sudden closure of the jaw when it is sharply struck downward. The reflex arc is in the fifth cranial nucleus It tests the masticatory nucleus of the fifth pair of cranial nerves.

Masseter Reflex: Closure of the jaw occurs when the insertion of the masseter muscle near the zygomaticus is struck. It tests the masticatory nucleus of the fifth cranial nerves. It is exaggerated in tetany (SEE: Chvostek's Sign, p. 791).

Paradoxical Reflex (Westphal): This consists of contraction of the tibialis instead of the calf muscles when the test for ankle clonus is being made, and also a contraction of the flexors instead of the extensors of the thigh upon an attempt to elicit the knee jerk with the patient in the dorsal position, when the patient sits up the normal reflex is elicited. It is found in various spinal cord diseases, in multiple sclerosis, and in paralysis agitans.

Defense Reflexes and Reflexes of Spinal Automatism: These result in special movements of retraction of the lower extremity which succeed excitation of the skin of the foot or forced flexion



Fig 17—Technic for eliciting the triceps reflex.

of the toes. The foot then flexes on the leg, the leg on the thigh, the thigh on the pelvis The mechanism is unexplained They may occur in pyramidal tract affections, also in cases of flaccid paraplegia with areflexia, whether the sensibility is intact or not. They may

be observed at a relatively early stage of complete spinal section when the other reflexes are wanting. They may also be produced by stimulation of the skin of the leg, the thigh, and the trunk, although it is less easy to produce them thus than by stimulation of the distal part of the limb. Babinski proposed their use in determining the inferior limits of spinal tumors.

Closely associated with the tendon reflexes are the osteoperiosteal reflexes (SEE: p 840).

Visceral Reflexes: Reflexes control the activity of the various viscera; among those reflexes are the bladder (or vesical) and rectal reflexes These are concerned in the retention and in the evacuation of the contents of the bladder and of the rectum

Retention of urine or *the inability to retain it,* when not caused by nervousness or mechanical obstruction, usually indicates disease of the spinal cord. Sphincter paralysis with empty bladder and constant dribbling of urine is found in lesions of the lumbar enlargement.

Detrusor paralysis with distended bladder, and often with dribbling of urine is found in lesions above the lumbar enlargement.

Urination and defecation are reflex acts under the control of the higher centers. The removal of inhibitory influence of these centers will cause a loss of sphincter control with involuntary urination and defecation as a consequence.

The rectal and visceral centers are in the lower lumbar and upper sacral segments.

Loss of sphincter control is seen in Lesions of the pyramidal tract; transverse and diffuse myelitis, tabes dorsalis; dementia paralytica; deep coma due to any cause; and various forms of dementia.

Nerve Mechanism of Bladder, Rectum and Penis: The *vesicospinal center* is in the conus medullaris. To it run fibers of the hypogastric plexus. From it (efferent) run the branches from the lumbar roots which pass through the lumbar sympathetic and the vesical plexus to the sphincter of the bladder, and also the nervi erigentes from the second and third sacral nerves which enter into the formation of the hypogastric plexus and supply the bladder walls. The center from which the nerves to the sphincter emerge is under the control of the brain, which both inhibits and reenforces it. The center from which the nerves to the walls emerge is not connected with the higher centers.

In cerebral lesions where inhibition is lost, the bladder empties spontaneously when a certain degree of distention has been reached.

In spinal lesions that affect the vesical centers there is true incontinence, *i. e.,* filling of the bladder and an involuntary flow of urine through the relaxed sphincter

In transverse spinal lesions above the spinal vesical centers there is loss of the sense of fullness of the bladder. Here the sphincter remains closed, and the urine is lost in drops (paradoxical incontinence). In certain favorable cases of paradoxical incontinence, a state of reflex micturition, under the influence of reflex stimulation from the lower extremities and the trunk, is established independently of the will.

The *anorectal mechanism* is analogous to the vesical. The centers are situated in the third and fourth sacral segments. The analogy is modified to some extent by the fact that under

arm rests upon the examiner's arm, palm upward, the elbow joint is supported by the examiner's hand so that the thumb rests in the cubital fossa. With a pleximeter hammer in the free hand, the examiner taps his own thumb smartly. The reflex arc is in the fifth and sixth cervical segments.



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Masseter Reflex: This has already been mentioned. It involves the motor nucleus of the fifth nerve (SEE: p. 838).

Oculocardiac Reflex: Compression of the eyeballs for more than five or ten seconds may produce modifications of the frequency of the cardiac rhythm and sometimes of the blood pressure. In the normal subject there may be a retardation of five or six beats a minute In pathologic cases the slowing may be more marked and there is also an appreciable lowering of blood pressure. In some cases there may be an increase rather than a decrease in pulse rate. It is a trigeminovagosympathetic reflex, and is supposed to be a test for vagotonia or for sympathicotonia, depending upon whether the vagus or the sympathetic nerve is the more irritable (decrease or increase in pulse rate respectively)

Carotid Sinus Reflex: Pressure upon the carotid sinus will elicit the carotid sinus syndrome which is characterized by attacks of syncope, vertigo, weakness and convulsions, either general or epileptiform The pulse is generally slow.

Various Other Reflexes and Signs: ***Digital Reflex or Hoffmann's Sign:*** A sudden nipping of the nail of the middle or ring fingers produces flexion of the terminal phalanx of the thumb and index finger and of the second and third phalanx of other fingers This is seen in pyramidal tract diseases affecting the upper extremities.

Magnus and de Kleijn Tonic Neck Reflex: This consists of extension of both ipsilateral limbs, or one, or part of a limb, and increase of tonus on the side to which the chin is turned when the head is rotated to the side, and flexion with loss of tonus on the side to which the occiput points (Wechsler[1]) This sign is found in decerebrate rigidity and in many severe cases of tuberculous meningitis of infants and young children.

Brudzinski's Signs: Contralateral Reflex. In meningitis when one lower extremity is flexed at the knee, there is flexion of the other lower extremity at the knee.

Neck Sign. In meningitis, when the neck of the patient is bent forward, flexion movements of the ankle, knee, hip and sometimes of the elbows are produced This is what is usually meant when the Brudzinski sign is referred to

Symphysis Sign· In meningitis, pressure on the symphysis by the physician's finger causes contraction of the lower extremities

Cheek Sign· In meningitis, pressure on both cheeks just below the malar bone causes raising of both arms with flexion of the elbow joints.

Babinski's Ear Reflex: When a galvanic electrode is placed near the ear of a patient suffering with disease of the middle or internal ears, the head will be inclined to the diseased side when the galvanic current is closed, and not, as in normal subjects, always toward the positive pole. This is also known as *vertigo voltaique pathologique,* or, at least, represents this condition Usually what is called in America the Babinski sign is the reflex extension of the toes, especially of the great toe, when the sole of the foot is irritated, in pyramidal tract affections

Paradoxic Pupillary Reflex: Dilatation of the pupil may occur on exposure to light as is sometimes seen in tabes and in general paralysis

Chaddock's Reflex: Stimulation below the external malleolus produces ex-

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[1] Wechsler, I S · Textbook of Clinical Neurology, 4th Ed., W B Saunders Co, 1940

ordinary conditions the feces are solid. In transverse lesions above the third sacral segment the tone of the sphincter may be maintained, but in spite of this, in consequence of the interruption of centripetal paths, the need for defecation *is not* felt. In certain patients with disturbances of the anorectal mechanism there is retention of feces; in others *especially those with* soft or liquid stools, diarrhea occurs. The subject is somewhat more complex than is that of retention and incontinence of urine.

The center for *erection of the penis is situated in* the second and third sacral segments. The centers for erection and ejaculation seem to be more or less independent.

Alterations of the conus medullaris and of the cauda equina may cause absence of erection and of ejaculation.

A state of priapism may occur in young individuals, with lesions of the cord above the lumbar region, or there may be a state of turgescence of the corpora cavernosa without erection

Vasomotor Reflexes: The stroking of an area of skin gives rise to a primary pallor (vasoconstriction), which is followed by a redness (paralytic vasodilation). Persistence of the redness is known as dermatographism (red). If the whiteness continues (say, as a line due to stroking), the persistence of the reflex is known as Sergent's white line, supposed by Sergent to indicate an insufficiency of suprarenal gland secretion. It is also found in other diseases.

Sweat Secretion: Sweat secretion is under the control of the nervous system, especially of the sympathetic system, by means of true secretory fibers that supply the sweat glands.

Normal Osteoperiosteal Reflexes: ***Radial Reflex:*** This consists in contraction of the supinator longus, biceps, brachialis anticus muscles when the styloid process of the radius is percussed

Ulnar Reflex: Contraction of the pronator teres with a movement of pronation of the hand takes place when the styloid process of the ulna is percussed when the elbow is semiflexed and the hand is in slight supination (ulnopronator reflex of Marie and Barré).

Periosteal Reflexes of the Adductors: Contraction of the corresponding adductor muscles of the thigh occurs when the internal condyle of the femur is percussed. This is often associated with the knee jerk on account of the proximity of the spinal centers that govern these two reflexes.

Semitendinosus and Semimembranosus Periosteal Reflex: This is elicited by percussion of the external tuberosity of the tibia. Contraction of the semitendinosus muscles and semimembranosus muscles follows

Inversion of the Reflexes: In destructive lesions of the segments governing these various reflexes, inversion of these may be seen, *i. e*, there may be flexion instead of extension or *vice versa*.

Reflexes Involving Some Cranial Nerves: ***Corneal Reflex:*** This traverses the trigeminofacial reflex arc (nucleus of the seventh cranial nerve). It may be absent in hysteria, corneal anesthesia, very deep general anesthesia, and in profound coma. It consists of closure of the lids when the cornea is touched.

Pharyngeal Reflex: This traverses the ninth and tenth cranial nerves (nucleus ambiguus). It consists of movements of deglutition when the pharynx is touched.

includes *aphasia proper* and *agraphia* or the inability to express ideas in writing. *Sensory aphasia* consists of word blindness and of word deafness.

According to the classical scheme *motor aphasia* is due to a lesion of Broca's area at the foot of the third left frontal convolution, or of the fibers leading from it, or of fibers connecting it with the other speech centers.

Agraphia is due to lesions of the second frontal convolution just superior to Broca's area or the fibers leading from it.

Word blindness is due to lesions of the angular gyrus Word blindness should not be confused with cortical blindness in which objects as well as words are involved, or the cortical lesions affecting the region around the calcarine fissure in which (if they are unilateral), hemianopsia is present

Word deafness is due to lesions of the first temporal convolution Word deafness may be present as a part of general bilateral auditory nerve deafness and of cortical deafness in which no sounds at all are heard.

In aphasia the lesion is in the left hemisphere in right-handed people

Types of Aphasia: A few of the characteristics distinguishing the various forms of aphasia are as follows.

Subcortical or *pure motor aphasia* is distinguished from the motor aphasia of Broca (due to lesions of Broca's convolution), in that, in the latter, inner speech is gravely affected Spoken and written language are understood in both varieties Writing may be more or less affected in Broca's form

In *total sensory aphasia* there are combined affections of the auditory and the visual centers for speech.

In *pure verbal deafness* (subcortical lesion), internal language is conserved so that the patient speaks, reads, and writes without paraphasia, paralexia or paragraphia.

In pure verbal blindness (subcortical lesion), internal language is conserved. The patient may speak without paraphasia, and may write spontaneously without paragraphia. It is sometimes associated with musical blindness and with right homonymous hemianopsia.

Optic Aphasia (Freund)· Here the use of an object is recognized by sight, but its name is not recalled unless sound, taste, and touch come to the aid. Even then the name is not always recalled This is really a variety of *agnosia,* which has been defined by Wilson as inability to recognize objects with conservation of primary sense perception

Transcortical Motor Aphasia. Spontaneous speech is lost. Here words can be repeated, print can be read aloud, and letters can be written from copy or dictation

Transcortical Sensory Aphasia: Here the power of comprehending written and spoken language is lost. There is spontaneous speech (sometimes with paragraphia). The patient can repeat words without comprehending their meaning and can write to dictation or from copy without understanding what he writes Some authors regard this as "psychic blindness" or "psychic deafness." Other authors apply these terms to agnosic disturbances These agnosic disturbances are, however, transcortical (Wilson).

Marie's View of Aphasia: Marie recognized three different syndromes in aphasia: 1. Sensory aphasia; 2. Anarthria, corresponding to pure motor aphasia (see below), and 3 Aphasia of

tension of the great toe. It occurs in lesions of the pyramidal tract and hence is to be classed with the Babinski, Gordon, and Oppenheim toe reflexes.

Conditioned Reflex: By this is meant a reflex that continues to be excited by kinds or nature of stimuli different from those of the original stimuli but which occurred originally in association with the original stimuli; thus salivary juice may be secreted in a dog on the ringing of a bell alone, if the bell had been rung when the dog actually took or saw food a certain number of times.

Croft's Reflex: Stroking with a blunt point upward over the dorsal surface of the ankle, the leg being horizontal and the muscles relaxed, causes dorsal extension of the great toe in cases of organic disease of the pyramidal tract

Gordon's Finger Reflex: Pressure on the radial side of the pisiform bone causes dorsal flexion and spreading of the fingers; this is seen in hemiplegia.

Mass Reflex (SEE: Defense Reflexes and Reflexes of Spinal Automatism, p. 835): A reflex may be exhibited by the entire area controlled by the portion of the spinal cord which has been injured. For example, if the spinal cord be transected, after the reflexes have been regained, they will be found to have lost their specific character, and afferent stimuli occasion diffuse and widespread motor reactions.

Upper Motor Neuron (Central) Reflex: Destruction of the pyramidal tract by a lesion in the internal capsule, by progressive primary destruction of the lateral columns or by section of the spinal cord will cause the upper motor neuron reflex. This consists of hyperactive deep tendon reflexes, spasticity and incoordination of the muscles with increased tonus, but with normal electrical reaction and abnormal reflexes such as positive clonus and Babinski reflexes. This is explainable by the fact that the motor ganglion cells of the anterior horn and their motor nerves remain unimpaired, but are cut off from the inhibiting and regulating influence of the cerebral centers by the lesion in the pyramidal tract.

Lower Motor Neuron (Peripheral) Reflex: Destruction of a lower motor neuron causes flaccidity, loss of motor function (complete paralysis), atrophy and electrical reaction of degeneration in the affected muscles. The skin and tendon reflexes are lost due to destruction of the motor limb of the reflex. At times the meningeal type of reflexes may be elicited, *viz*, Kernig's and Brudzinski's signs

Westphal's Pupillary Reflex: Contraction of the pupil may be associated with closure or attempted closure of the eye

For other signs see pupillary reflexes (p 182). Signs in tetany (p 791), and signs in exophthalmic goiter, see p. 778.

Examination of Disturbances of the Speech Centers

Alterations of language consist in inability of expression due either to paralysis of the muscles concerned in articulate speech, which may occur in subcortical or nuclear lesions (dysarthria), or in aphasia.

Aphasia: This implies the inability to express oneself by articulate speech, by signs or by writing, as well as the inability to comprehend spoken or written language by one who has no defects of the peripheral organs and is not unfamiliar with the language spoken or written by the examiner. *Motor aphasia*

Wilson regards tremor as due to disease of the old or extrapyramidal motor system.

Myoclonus: This is experienced in clonic contractions, not epileptiform, which affect the muscles of the limbs and of the trunk especially. The movements are rapid, fulminating, often preceded, accompanied, or succeeded by fibrillary contractions. They cease during sleep and are usually bilateral They occur chiefly in encephalitis and in paramyoclonus multiplex.

Tics: These are tonic or clonic, more or less easily imitated, coördinated gross movements, associated with poor power of the patient to coöperate and sometimes with the repetition of words or phrases. The tics may be symptomatic or regarded as a disease *sui generis.*

Nystagmus: This consists of rapid associated conjugate movements of the eyeballs, and may be either static or dynamic. There are two components of a nystagmus; a slow movement in one direction, followed by a rapid movement in another direction, and toward the right or toward the left. There may also be vertical and rotary nystagmus In nystagmus, the associated movements of the eye are involved It may occur in miners, in albinos, or congenitally, in cerebellar affections, in vestibular affections, in Friedreich's ataxia, and in multiple sclerosis. Nystagmus usually points to cerebellar or labyrinthine disease According to Bing, nystagmoid movements backward and forward may occur.

Apraxia: This is the inability to execute purposeful movements.

Types of Apraxia (1) Motor apraxia; when the patient is unable to execute movements or commands.

(2) Idiomotor apraxia; when the patient is unable to imitate movements performed in his presence.

(3) Parapraxia; when the patient executes movements other than those commanded him.

(4) Intentional perseveration of Liepmann; when the patient executes one movement correctly as ordered, but when told to perform another kind of movement continues repeating the first.

(5) Clonic perseveration of Liepmann; when the patient continues to perform an action or a motion for some time after being told to stop.

Left-sided apraxia is sometimes produced by lesions of the corpus callosum; it has also been noted, according to Potts, in lesions of the left frontal lobe and of the left parietal lobe

Muscle Tonus (*muscle tone*): Muscle tone is defined as a state of reflex contraction which is concerned with maintaining position and posture. This is regulated by impulses that proceed from the anterior horn cells. These cells are themselves subjected to tone-regulating impulses, which travel along the descending tracts from the brain. The motor tracts accessory to the pyramidal tracts are important factors in this tone-regulating property It is probable that the cerebellum also exercises a regulatory function on tone

Reflex tone depends mainly on afferent impulses coming from the sense organs in the muscles themselves and, to a less extent, on impulses from the vestibular apparatus and the eyes. Wright[1] states that there is no essential difference between the contraction which maintains tone and that which executes movements "Muscle tone is probably due to a slow asynchronous discharge from

[1] Wright, S Applied Physiology, 6th Ed., page 63 Oxford University Press.

Imitative Synkinesias: These occur on the sound side when a movement is executed or tends to be executed on a paralyzed side. According to S. A. K. Wilson, these may occur typically in Parkinson's disease.

Synkinesias of Coördination: In these it is possible to execute synergically a given movement, which voluntarily and when isolated cannot be executed by the patient, and it is impossible to inhibit this movement when the synergists act. An example of such a movement is the *tibialis phenomenon of Strümpell* Here the foot cannot voluntarily be flexed dorsally on the leg, in hemiplegia and in monoplegia of the lower extremity; nevertheless the foot draws up in spite of efforts on the part of the patient to prevent its extension, if the patient flexes his thigh on his pelvis and his leg on his thigh. According to Marie, this type of synkinesia is the expression of the automatism of lower centers.

Adiadokokinesis: This is the inability to arrest one motor impulse and substitute for it one that is diametrically opposite.

Athetosis: These are slow vermicular bizarre movements of the extremities, especially of their distal portions Their existence has been ascribed to lesions of various of the basal ganglia This view is opposed by Wilson, who regards them as due to cortical factors.

Choreic Movements: These movements are described by S. A K. Wilson as subjectively purposeful but objectively purposeless Each fresh movement appears to be directed to an end which is never attained. They are brief and unsustained. According to Wilson, both chorea and athetosis are involuntary movements of the new motor or cortical system rather than of the old or extrapyramidal system.

Spasm: This is due to an irritative condition. It consists of more or less prolonged involuntary muscular contractions. When the spasm is prolonged, it is known as *tonic*, when it is intermittent, consisting of a series of muscular jerks, it is known as *clonic*. Spasm is associated with convulsions, with epilepsy, and with tetany and tetanus It may persist during sleep.

Tremors: These are involuntary, more or less rapid, oscillatory movements. They may be classified as: (*a*) Static tremors, seen in a state of repose, which diminish or cease on voluntary movement of the part (paralysis agitans, parkinsonian form of encephalitis lethargica); (*b*) dynamic or kinetic tremors, the intention tremors of disseminated sclerosis; (*c*) tremors seen in repose and on attempted movements, hereditary tremors, hysteric tremor.

All pathologic tremors are independent of the will They can, however, be produced or simulated voluntarily. Emotions tend to exaggerate them. They usually, but not always, cease during sleep. They vary much in rapidity, in amplitude, and in location They occur in toxic conditions (abuse of alcohol and of other drugs), in general paralysis and in old age. As is well known, tremors are characteristic of hyperthyroidism and Graves' disease. They occur in neurasthenia and in the functional neuroses generally. They may follow apoplexy, be associated with degeneration of certain parts of the cerebellum, dyssynergia cerebellaris progressiva of Hunt, and with pseudosclerosis, lenticular degeneration, multiple sclerosis, cerebrospinal syphilis; the centers and tracts governing muscular tonus are here involved.

Dissociation of various forms of sensibility are as follows:

Syringomyelic: Tactile and deep sensibility are retained, thermic and pain sensibility are abolished (over a portion of the body).

Tabetic: Relative conservation of the thermic and pain sensibility exist, together with abolition (at least in part) of the tactile sense and of the deep sensibility.

Cutaneous Deep: Abolition or diminution of the pressure, osseous, and musculoarticular sensibility occurs, with conservation of tactile, thermic, and pain sensibility.

Anesthesia Dolorosa. Painfulness of a part is seen, as of a limb or of a half of the body, associated with anesthesia of that part. Seen in thalamic lesions

Hyperesthesia This symptom is seen in a variety of conditions.

Dysthesia: (a) *Retardation of sensation*; (b) *fusion of the sensations* due to successive stimuli (in a prolonged sensation); (c) *addition of sensations*, perception of sensation only after repeated excitations, (d) *errors of localization*; (e) *perceptions of the first only* of a series of excitations; (f) *disappearance of the sensation* during a prolonged stimulation; (g) *polyesthesia*, several sensations felt when the stimulus is single, (h) *synalgia*, painful sensation far from the point excited; (i) *allochiria*, perception of the sensation at a symmetrical point of the body; (j) *metamorphosis of sensations*, false interpretation of a given stimulus

Subjective Sensations: Pain is found especially in neuralgia, neuritis, and radiculitis, and in diseases of the central nervous system in which the sensory tracts are involved

Causalgia: A spontaneous pain, especially when it is burning in character, associated with anesthesia or hypesthesia in the sensory distribution of a given nerve is termed causalgia It seems to be bound up with lesions of the nervi nervorum.

Paresthesia: Sensations of formication, tingling, and the like are found in central and in peripheral lesions.

Pseudomyelia Paresthetica (Bechterew): A false sensation of movement in a paralyzed limb sometimes is seen The converse of this may occur, *i e.*, a *sensation of lack of movement when the* limb is really moving. (Mingazzini, quoted by Mattirolo.)

Disturbances of Equilibrium and Orientation

The principal organs of coordination of equilibrium and of orientation are the cerebellum and the cerebrum.

Ataxia (loss of coordination): ***Static Ataxia:*** Slow and wide oscillations in a limb when an attempt is made to keep it at rest, in the trunk when the patient is seated, in the body when the patient is on his feet reveal static ataxia

Dynamic Ataxia: *Incoordination in the execution of a movement suggests* dynamic ataxia

Tabetic Ataxia: This occurs when there are lesions of the first order of sensory neurons (neuritis or tabes dorsalis); in lesions of the second order of sensory neurons (bulbar and pontine ataxia); in lesions of the third order of sensory neurons, cerebral ataxia.

Cerebellar Ataxia: This is found in lesions of the cerebellum; lesions of the afferent and efferent fibers of the cerebellar system, central or peripheral lesions of the vestibular apparatus.

anterior horn cells producing a partial tetanus which is economical and can be maintained. Movement is due to a more rapid synchronous discharge which gives rise to a more powerful tetanus but of relatively short duration."

Reaction of Degeneration: When a faradic or galvanic current is applied to a normal nerve or muscle, a sharp contraction will occur while the current is passing A diseased muscle will not readily respond to a faradic current, but will respond to the positive pole of the galvanic current. A diseased nerve will not respond to either pole of any current

When the cathode (negative pole) is placed over a certain point of a normal muscle (motor point) and the other pole over the spine, a strong contraction occurs when the circuit is closed or broken. When the anode (positive pole) is placed over the point, the contraction is much less In neither case is there any contraction when the current is passing. The reaction of degeneration consists in the reversal of these phenomena, at least the so-called "aerial change" as expressing degeneration does. Complete reactions of degeneration include modal changes and loss of reactions to the faradic current.

The following formulae express the electrical reactions:

NORMAL MUSCLE

AnClC is less than CaClC
(Anodal [positive] closing contraction is less than cathodal [negative] closing contraction)

AnOC is greater than CaOC
(Anodal opening contraction is greater than cathodal opening contraction.)

MUSCLES IN THE FIRST STAGE OF DEGENERATION

AnClC equals CaClC
(Anodal closing contraction equals cathodal closing contraction.)

AnOC equals CaOC
(Anodal opening contraction equals cathodal opening contraction.)

MUSCLES IN ADVANCED STAGE OF DEGENERATION

AnClC is greater than CaClC
(Anodal closing contraction is greater than cathodal closing contraction)

AnOC is less than CaOC
(Anodal opening contraction is less than cathodal opening contraction.)

Reaction of degeneration is observed in advanced acute and chronic poliomyelitis, acute central myelitis, progressive muscular atrophy, and in severe peripheral neuritis after compression of a nerve. This reaction indicates that the trophic cells in the anterior gray horns of the cord have been destroyed, or that the efferent fibers from these cells have degenerated or that there has been extensive atrophy of the muscle

Vermicular responses of the muscles to electrical stimulation are considered signs of degeneration (the so-called "modal change").

The *myotonic reaction* is involuntary persistence of the contraction after faradic stimulation of the muscle. It is seen in myotonia congenita and myotonic dystrophy

The *myasthenic reaction* (of Jolly) is the rapid exhaustion of the responses to faradic stimulation of the muscle and nerves. It is seen typically in myasthenia gravis, although it may not occur here, and has been reported as having been found sporadically in other conditions.

Reactions of Sensibility: The following forms of sensibility are to be tested: Tactile; pressure; thermic; pain; musculoarticular; osseous (use of a tuning fork on the bones), and stereognostic

in extracerebellar affections that involve the function of the cerebellum (SEE: p. 814).

Cerebellar Vertigo: This is typical rotary systematic vertigo, which is present in the erect and recumbent postures. It is accompanied by vomiting, sweating, and syncope and seems to vary with the intracranial pressure. It is found also in lesions of the pathways that unite the vestibular nerve with the cerebellum, in which case the systematic vertigo would tend toward the extracerebellar type.

Labyrinthine Vertigo (Ménière): This occurs in lesions of the vestibular apparatus, including Dieters's nucleus It is essentially paroxysmal It is also systematic, that is, the rotation of the organism or of surrounding objects is always in a given direction.

Vertigo also occurs in circulatory diseases of the brain, and in cerebral tumors (nonsystematic); in lesions of the bulb and the pons (perhaps systematic); in paralysis and contractions of the eye muscles with strabismus and diplopia (nonsystematic), in inhalations of fumes; following painful impressions of the nasal and laryngeal mucous membranes; in diseases of the gastrointestinal tract and liver, and in various toxic states.

Lateropulsion and Lateral Deviation of the Body: This is observed in cerebellar lesions (lesion on the same side as the lateropulsion and lateral deviation). They occur toward the same side also in lesions of the inferior cerebellar peduncles, and toward the opposite side in lesions of the superior cerebellar peduncles.

Gaits: Abnormal gaits are associated with disturbances of equilibrium. The *tabetic gait* is characterized by widespread legs, goose-step, and concave knee. Lateropulsion has already been mentioned. In the gait in *paralysis agitans* the patient tends to run after his center of gravity. The form of *encephalitis lethargica* that simulates paralysis agitans may be associated with a slow awkward gait; in some of these the gait is difficult to distinguish from that of true paralysis agitans. In *multiple neuritis* affecting both legs the gait resembles that of a high-stepping horse; hence, the term *"steppage gait."*

The gait of *hemiplegia* may be readily recognized, as well as the so-called *"crossed leg"* progression of *infantile palsy* The gait of *dysbasia lordotica progressiva* (torsion spasm) is peculiar and has been called the *"dromedary gait."* The gait of *Huntington's chorea* is also peculiar, consisting of a few normal paces, then a long slow pace, and then one or two hops (SEE. p 120).

The mixed tabocerebellar type of ataxia is found in lesions of the cerebellum and of the spinocerebellar tracts associated with those of the primary sensory neurons (Friedreich's ataxia).

Tabetic ataxia is both static and dynamic. When the lower limbs are affected, there is the characteristic goose-step or tabetic gait. During walking, when the trunk is affected, there are oscillatory movements of the body. The dynamic ataxia of the upper extremities is manifested in all their movements, especially in the finer movements. Incoordinate and excessive movements of the face may be observed when the patient talks, laughs, or weeps. Static ataxia may be demonstrated by asking the patient to raise his arms or his legs while his trunk remains supine; or the ataxia of the trunk may be demonstrated by the sway of the body when the patient closes his eyes, his feet being together, standing posture (*Romberg's sign*).

Cerebellar ataxia may be demonstrated by lateral and anteroposterior movements of the body while attempting to maintain equilibrium, and by staggering or zigzag movements, more to one side than to the other. Difficulty is found in the grasping of objects. Closure of the eyes has little or no effect on the unsteady station. This is seen in lesions of the cerebellum and its pathways, and in lesions of the vestibular apparatus.

The tabetocerebellar type partakes of the characteristics of both the tabetic and the cerebellar forms of ataxia.

Asynergia: This is characterized by a want of harmony between muscle groups in the execution of a movement; thus, in walking, a lower extremity may be advanced while the trunk is unprepared for the movement; this is decomposition of movements that ordinarily occur simultaneously; *i.e.*, the individual movements occur in serial order instead of together. This is present in affections of the cerebellum.

Adiadokokinesis: This occurs in cerebellar affections Rapid antagonistic movements, for instance, those of pronation and supination of the hands, cannot be carried out repeatedly with accuracy.

Dysmetria: This also occurs in cerebellar affections. The movements are rapid and brusque, as if the degree of force necessary to execute them were misjudged.

Past Pointing: This is the failure of the index finger of the patient to touch an object when he attempts to touch it, the finger passing the object with more or less latitude. The patient is asked to touch the tip of his nose with his index finger after having his arm at full extension or to touch the tips of the index finger of both hands after the hands have been far apart. It is best carried out as a test when the patient's eyes are closed. Past pointing may occur spontaneously in conditions associated with ataxia, especially in cerebellar and vestibular nerve conditions, or may vary from normal past-pointing reactions when the Bárány tests are carried out in a study of these conditions.

Vertigo (sensation of loss of equilibrium): Vertigo is a sensation in which objects and the body of the patient himself seem to be in space while they are really at rest. This may occur in a given direction (systematic). Sometimes the body and the objects seem to be turning in the same direction; according to Stewart and Holmes, this is found in cerebellar affections proper. Sometimes the body seems to turn in an opposite direction to that of the objects; according to Stewart and Holmes, this is found

hypothenar eminences, interossei, and lumbricals.

(*b*) Anesthesia of the ulnar side of the forearm, but not of the part of the (upper) *arm that is innervated by the* second dorsal root

(*c*) Sympathetic oculopupillary paralysis

Lesions of the Brachial Plexus: Partial Lesions:

1. ***Syndrome of the Outer Cord:*** Paralysis of the muscles innervated by the musculocutaneous nerve and by the external head of the median nerve, *i. e.*, biceps, coracobrachialis, brachialis anticus, palmaris longus, pronator teres, and the flexors and the opponent of the thumb.

2. ***Syndrome of the Inner Cord:*** Paralysis of the muscles innervated by the ulnar and by a part of those innervated by the median (internal head). These latter are the flexors of the fingers.

3. ***Syndrome of the Posterior Cord:*** Paralysis of the muscles innervated by the circumflex nerve and by the musculospiral nerve.

The alterations in sensibility in lesions of the brachial plexus are neither radicular nor do they follow the anesthesias of wounds of the peripheral nerves

Lesions or injury to the nerve supplying the chest muscles, such as the rhomboids, the serratus magnus muscle, the suprascapular muscle, the great pectoralis muscle, the latissimus dorsi muscle, will cause them to be paralyzed

Lesions of the Circumflex Nerve: These result in paralysis of the teres minor and the deltoid muscles and anesthesia over the insertion of the deltoid muscle

Lesion of the Musculospiral Nerve: Wrist Drop: The following movements are lost: (*a*) Extension of the forearm on the arm (paralysis of the triceps *muscle*); (*b*) *supination of the forearm* (supinators); (*c*) extension of the hand at the wrist (radial and posterior ulnar muscles); (*d*) extension of the first phalanges on their metacarpal bones (extensors of the fingers).

Anesthesia occurs along the cutaneous course of the nerve.

Lesions of the Median Nerve: Lesions of this nerve produce the following paralysis of movement: (*a*) Flexion of the hand on the forearm; (*b*) pronation of the forearm; (*c*) flexion of the thumb, index finger, and middle finger, (*d*) apposition of the thumb.

The characteristic is anesthesia of the thumb, the two adjoining fingers and the half of the next on their palmar surface, the corresponding part of the palm to the wrist, and, on the back of the hand, of the two end phalanges of the two and a half fingers next to the thumb

Lesions of the Ulnar Nerve: The following paralysis is produced. (*a*) Extension of the last two phalanges of the ring and the little finger; (*b*) adduction of the thumb, in part compensated by the action of the opponents; (*c*) spreading and approximating of the fingers; (*d*) adduction and apposition of the little finger, (*e*) flexion of the first phalanx of the four fingers.

Anesthesia occurs in the ulnar nerve distribution.

Lesions of the Musculocutaneous Nerve: These result in loss of flexion of the forearm on the arm; anesthesia in the arm of cutaneous distribution of the nerve.

CHAPTER XXVIII

Diseases of the Nervous System

Diseases of the nervous system are of two types, organic and functional. Organic nervous diseases occur as the result of definite lesions in some part of the nervous system which interfere with either perception, conduction or innervation of muscles, glands or other structures of the body and affect their specific functions. These lesions are identifiable by tracing the primary defects to the physiologic nerve center. Such lesions may be due to infections, degenerations, inflammation, tumors, hemorrhage or other destructive processes.

Functional nervous diseases occur in the absence of any discoverable organic lesion. The principal defects are associated with disturbance of the orderly mental processes, and are termed neuroses, psychoses and psychoneuroses

Organic Diseases of the Nervous System

Organic diseases of the nervous system are studied by means of physical examination, by examination of the spinal fluid and the blood, by x-rays and by special tests.

Lesions of the Peripheral Nerves

Paralysis of the Phrenic Nerves: ***Bilateral:*** The auxiliary muscles of inspiration come into play The patient is dyspneic. Both inspiration and expiration are difficult.

Unilateral: In unilateral paralysis, Litten's diaphragmatic phenomenon is wanting on the paralyzed side.

Total Radicular Paralysis of the Brachial Plexus: This causes: (*a*) Flaccid paralysis of all the muscles of the upper extremity and of the shoulder girdle.

(*b*) Complete anesthesia of this extremity with the exception of the inner surface of the arm.

(*c*) Sympathetic oculopupillary paralysis by reason of the anastomosis of the plexus with the communicating branch of the first dorsal nerve

Superior Radicular Paralysis (5th and 6th cervical roots): *Erb's* palsy is manifested by: (*a*) Paralysis of the deltoid, biceps, brachialis anticus, and long supinator muscles. At times also the levator anguli scapulae, the rhomboids, infraspinatus, supraspinatus, and *serratus magnus may become paralyzed.*

(*b*) Anesthesia of the external and radial side of the forearm.

(*c*) Triceps reflex preserved; radial periosteal reflex abolished.

Medial Radicular Paralysis (*7th* cervical root): This causes: (*a*) Paralysis of the extensor communis digitorum, long and short extensors of the thumb, extensor proprius of the index finger and extensor proprius of the little finger, long abductor of the thumb, the two extensors (carpi radialis, the extensor carpi ulnaris), *partial reaction of degeneration in the paralyzed muscles*

(*b*) Hypesthesia in a longitudinal zone on the posterior surface of the forearm.

Inferior Radicular Paralysis (8th cervical and 1st dorsal *roots*): *Klumpke's palsy* results in: (*a*) Paralysis of the flexors of the fingers, flexor carpi ulnaris, small muscles of *the thenar* and

Lesions of the Small Sciatic Nerve: These produce a flaccidity of the buttock on the affected side with some difficulty in extension of the thigh, as in ascending stairs. Unilateral paralysis of the gluteus muscle gives rise to Trendelenburg's symptom, an inclination of the pelvis toward the sound side when the patient stands on the affected leg There is cutaneous anesthesia

Lesions of the Great Sciatic Nerve: Total paralysis causes paralysis and atrophy of the flexor muscles of the leg and the thigh, and paralysis and atrophy of all the muscles of the leg and foot Drop foot and steppage gait are present, on the affected side. There is anesthesia in the cutaneous distribution

Lesions of the Internal Popliteal Nerve: These produce paralysis of flexion and of adduction of the toes, of adduction and abduction of the toes, of rotation inward and adduction of the foot, of plantar flexion and of lowering of the ball of the foot There is anesthesia in the cutaneous distribution

Lesions of the External Popliteal Nerve: These produce paralysis of dorsal flexion and adduction of the foot, of rotation of the ball of the foot outward and of raising of the external border of the foot, of extension of the toes There is cutaneous anesthesia.

Neuritis: *Alcoholic neuritis* may affect all extremities, but has a predilection for the external popliteal nerve In *Korsakoff's psychosis*, usually alcoholic, there are in addition to neuritides, amnesia for recent events and memory gaps with a tendency to confabulation, intellectual weakness, delirium, hallucinations and illusions *Lead palsy* is largely confined to the upper extremities, is often of the lower arm type (wrist drop) with relative immunity of the nerve to the supinator longus muscle There may be the concomitant signs of lead poisoning *Arsenical neuritis* is usually confined to the distal parts of the extremities, and is apt to be associated with skin lesions. *Diabetic polyneuritis* prefers the domain of the anterior crural, obturator, and the peroneal nerves. *Diphtheritic paralysis* largely affects the palate, the pharynx, perhaps in certain cases the heart, and sometimes the eye muscles, through their nerves. *Beriberi* and *leprosy* may be causes of polyneuritis.

The *Guillian-Barré syndrome* is a radiculoneuritis. It consists of flaccid paralysis with paresthesias and myalgia, and it is progressive for several weeks. In the young, recovery is complete; in the old, the prognosis is doubtful The spinal fluid may be xanthochromic and contain excess protein with a normal cell count

Neuralgia: This is a symptom characterized by paroxysmal attacks of pain in the cutaneous distribution of a peripheral sensory nerve or its branches It may be caused by neuritis, pressure on a nerve trunk, or changes in the root ganglia, or in the nutrition of a nerve due to altered blood and nerve supply or to toxins. Sciatic neuralgia may resemble true sciatica In true sciatica, Lasègue's sign is positive, that is the inability to raise the extended lower extremity on the pelvis without producing popliteal pain Achilles' reflex is absent or weak

Pain in the abdominal wall, aggravated by superficial palpation or pinching, may be due to intercostal neuralgia

Lesions of the Cranial Nerves

Lesions of the First Cranial Nerve: These lesions include *anosmia* (loss of sense of smell), *hyposmia* (impaired sense of smell) and *hyperosmia* (acute

Lesions of the Trunks of the Lumbar Plexus: ***Lesions of the first and second lumbar trunks*** produce weakness of the psoas, quadratus lumborum, transverse abdominal and quadriceps femoris muscles, with anesthesia over the upper anterior part of the thigh and the external surface of the buttocks.

Lesions of the third and fourth lumbar trunks produce paralysis of the muscles applied by the anterior crural nerve and the obturator nerve, and weakness of the glutei, tensor fascia lata, semitendinosus, and other muscles supplied by the fourth lumbar trunk. In the leg the anterior tibial muscle is paralyzed or weakened The anesthesia covers the lower external surface of the thigh and the internal surface of the leg and the foot.

Lesions of the external cutaneous nerve determine the condition known as meralgia paresthetica.

Lesions of the anterior crural nerve produce paralysis of flexion of the thigh, of extension of the leg, and of rotation of the leg outward. There is anesthesia in the cutaneous distribution.

Lesions of the obturator nerve produce paralysis of adduction of the lower extremity, of approximation and crossing of the thighs, with anesthesia in the cutaneous distribution of the nerve

Lesions of the Sacral Plexus: These are sometimes produced by union of the fifth lumbar vertebra with the sacrum. Various neuralgias and neuritides are produced, which are associated with the bone and joint changes and which have been called Bertolotti's syndrome.

Lesions of the first and second sacral trunks produce, generally, paralysis of the muscles of the leg except the tibialis anticus, and also, generally, paralysis of the muscle of the thigh supplied by these trunks, and of the foot. There is anesthesia in the cutaneous distribution of these trunks.

Lesions of the third and fourth sacral trunks, if bilateral, produce a syndrome similar to that produced by lesions of the conus medullaris.

Lesions of the Cauda Equina: *Pain* in the perineum, and down the back and front of the thighs and legs, or in the small of the back usually precedes *the physical signs* *Later there* develops weakness in the limb which progresses to *flaccid paralysis.* There is *impairment of all forms of sensation* in the affected roots and of *deep reflexes; both ankle* and *knee* jerks are lost. There may be radicular distribution of anesthesia in the perineum, on' the buttocks and in the lower extremities; sometimes there may be associated motor paralysis (depending on the lesion) of the glutei and other nearby muscles, with atrophy and vesicle, rectal, and sexual disturbances. Paralysis of the bladder and rectum occur only when the lesion is in the sacral region. Often this is absent or it may occur as a late symptom. The symptoms are often asymmetrical or unilateral. Recovery may occur.

Lesions of the Conus Medullaris: These always cause *bilateral symptoms* Bladder and rectal disturbances occur early and are severe. Pain is not a prominent symptom; if present, it affects only the perineum and buttocks. Saddle anesthesia occurs early and there may be dissociation of sensation, that is, loss of pain and temperature alone Knee jerk reflex remains intact but ankle jerk is lost. Recovery does not occur. Occasionally there may be simultaneous involvement of both the cauda equina and the conus medullaris.

Lesions of the Small Sciatic Nerve: These produce a flaccidity of the buttock on the affected side with some difficulty in extension of the thigh, as in ascending stairs Unilateral paralysis of the gluteus muscle gives rise to Trendelenburg's symptom, an inclination of the pelvis *toward the sound side when the* patient stands on the affected leg There is cutaneous anesthesia.

Lesions of the Great Sciatic Nerve: Total paralysis causes paralysis and atrophy of the flexor muscles of the leg and the thigh, and paralysis and atrophy of all the muscles of the leg and foot. Drop foot and steppage gait are present, on the affected side. There is anesthesia in the cutaneous distribution

Lesions of the Internal Popliteal Nerve: These produce paralysis of flexion and of adduction of the toes, of adduction and abduction of the toes, of rotation inward and adduction of the foot, of plantar flexion and of lowering of the ball of the foot. There is anesthesia in the cutaneous distribution.

Lesions of the External Popliteal Nerve: These produce paralysis of dorsal flexion and adduction of the foot, of rotation of the ball of the foot outward and of raising of the external border of the foot, of extension of the toes There is cutaneous anesthesia.

Neuritis: *Alcoholic neuritis* may affect all extremities, but has a predilection for the external popliteal nerve In *Korsakoff's psychosis*, usually alcoholic, there are in addition to neuritides, amnesia for recent events and memory gaps with a tendency to confabulation, intellectual weakness, delirium, hallucinations and illusions. *Lead palsy* is largely confined to the upper extremities; is often of the lower arm type (wrist drop) with relative immunity of the nerve to the supinator longus muscle. There may be the concomitant signs of lead poisoning *Arsenical neuritis* is usually confined to the distal parts of the extremities, and is apt to be associated with skin lesions *Diabetic polyneuritis* prefers the domain of the anterior crural, obturator, and the peroneal nerves. *Diphtheritic paralysis* largely affects the palate, the pharynx, perhaps in certain cases the heart, and sometimes the eye muscles, through their nerves *Beriberi* and *leprosy* may be causes of polyneuritis.

The *Guillian-Barré syndrome* is a radiculoneuritis. It consists of flaccid paralysis with paresthesias and myalgia, and it is progressive for several weeks. In the young, recovery is complete, in the old, the prognosis is doubtful. The spinal fluid may be xanthochromic and contain excess protein with a normal cell count

Neuralgia: This is a symptom characterized by paroxysmal attacks of pain in the cutaneous distribution of a peripheral sensory nerve or its branches It may be caused by neuritis, pressure on a nerve trunk, or changes in the root ganglia, or in the nutrition of a nerve due to altered blood and nerve supply or to toxins Sciatic neuralgia may resemble true sciatica In true sciatica, Lasègue's sign is positive, that is the inability to raise the extended lower extremity on the pelvis without producing popliteal pain Achilles' reflex is absent or weak

Pain in the abdominal wall, aggravated by superficial palpation or pinching, may be due to intercostal neuralgia.

Lesions of the Cranial Nerves

Lesions of the First Cranial Nerve: These lesions include *anosmia* (loss of sense of smell), *hyposmia* (impaired sense of smell) and *hyperosmia* (acute

or exaggerated sense of smell). *Parosmia* is a perverted or false sense of smell.

These disturbances may occur in lesions of the olfactory centers, of the hippocampal region, of the horn of Ammon, and of the olfactory bulb and tract, also in syphilitic alterations, in basilar meningitis, tumors of the orbital lobe, and in hydrocephalus with compression of the olfactory tract. Anosmia may occur in tabes dorsalis (Klippel and Jullian). Alterations of the sense of smell may occur also in peripheral lesions of the olfactory paths and in ozena; also after the inhalation of irritant gases and in hysteria and toxic psychosis In testing the sense of smell, nonirritating substances, such as some of the essential oils (cloves, cinnamon), should be used

Lesions of the Second Cranial Nerve: The pupil reacts physiologically to light, to convergence, to accommodation, and to pain. It reacts to emotions The idea of a distant or dark object provokes a dilatation (Haab's reflex). *The Argyll-Robertson* pupil is one in which the pupil does not react to light but does to accommodation (seen in tabes and perhaps in other varieties of syphilis of the nervous system). The tabetic pupil is also a contracted pupil

The consensual reaction of the pupils to light should always be tested, *i. e.*, the pupil of a screened but observed eye should dilate and contract together with the pupil of the other eye.

In cases where the pupil does not react to light (tabes, oculomotor neuritis), contraction may be brought about by ordering the patient to close his eye while the physician exerts force with his fingers to keep the eye open. (Westphal-Galassi's phenomenon).

The examination of all cases of nervous disease should include an examination of the eye grounds and the fields of vision.

Destruction of the optic nerve anywhere from the retina to the chiasm produces loss of vision in the corresponding eye The reflex of the pupil to light is abolished, although the pupil reacts when the sound eye is illuminated. Partial destruction causes scotomata and gaps in the visual field.

The matter of optic neuritis and choked discs is discussed on p. 870

Lesions of the chiasm at the middle and lesions of the pituitary gland, because of pressure, will produce bitemporal hemianopsia.

A lesion of the chiasm at the side produces nasal hemianopsia Bilateral lesions (at the sides) produce binasal hemianopsia.

A lesion of the optic tract anterior to the primary optic centers produces homonymous hemianopsia of the field of vision opposite to the side of the lesion Illumination of the blind halves of the retina does not produce the reaction to light (hemiopic pupillary phenomenon) This is also true when the *primary optic center is involved.*

Lesions of the optic radiations produce homonymous hemianopsia with reaction to light when the blind halves are illuminated.

Lesions of the superior lip of the calcarine fissures produce quadrant anopia or anopsia in the inferior fields of vision opposite to the side of the lesion.

Lesions of the inferior lip of the calcarine fissure produce quadrant anopsia in the superior fields of vision opposite to the side of the lesion.

Lesions of the Third, Fourth, and Sixth Nerves: Total paralysis of the

third pair produces ptosis, deviation of the bulbs externally, of paralysis of the internal recti, of the superior recti, of inferior oblique, and of the inferior recti, paralytic mydriasis, loss of the reactions to light and to accommodation, and crossed diplopia.

Supranuclear lesions of the third nerve are associated with those of the sixth pair, and there is conjugate deviation of the eyes with paralysis of one external rectus and one internal rectus muscle.

Nuclear lesions usually cause complete external and incomplete internal paralysis; ordinarily, the pupillary reactions are preserved

Peripheral lesions are due to trauma, meningitis, tumors, infections, or toxic causes

In paralysis of the fourth nerve, the deviation is upward and inward; the false image occurs downward and outward

In paralysis of the sixth nerve, the deviation is inward; the false image outward Peripheral involvement of the sixth nerve, when associated with otitis media and with temporoparietal pain, is known as the *syndrome of Gradenigo.*

Associated Actions of the Eye Muscles: *Lateral Movements* Conjugate deviation of the eyes and the head: The lateral movements of the eyes are governed by a center at the foot of the second frontal convolution This sends pathways to the internal rectus muscle of the same side and to the external rectus muscle of the opposite side. There are paths joining the ocular nuclei The principal connecting pathway is the posterior longitudinal bundle which sends branches also to other cranial nerve nuclei The fibers from the cerebrum pass down through the knee of the internal capsule

In paralytic cortical lesions the eyes and the head are turned towards the side of the lesions. In spastic or convulsive deviation due to cortical lesions, the eye and the head are turned away from the side of the lesion. When the lesions are in the pons rather than in the cortex, the deviation in paralytic lesions is away from the side of the lesion and, in irritative lesions, toward the side of the lesion.

In paralysis of the *associated movements of elevation* the eyes cannot be elevated This may occur especially in tumors of the superior quadrigemina, which also may occasion paralysis of depression of the eyes.

Paralysis of convergence is often seen in Graves' disease (Moebius' sign).

Associated paralysis of the internal muscle of one side and the external muscle of the opposite side without deviation, is due to a lesion of the posterior longitudinal bundle on the side of the paralysis.

The subject of nystagmus has already been dealt with (SEE. p 847).

Lesions of the Fifth Cranial Nerve: *Destructive lesions of the motor part* produce paralysis of the muscles of mastication.

Lesions of the sensory part vary as to their symptomology according to the site of the trouble. Lesions of the gasserian ganglion give rise to anesthesia-like bands similar to those found in spinal lesions. This may occur in high syringomyelia and in syringobulbia The lesions may be the dissociated type. Degeneration of the descending root may occur in tabes; symptoms are produced similar to those just mentioned

Neuralgia and hyperesthesia may affect each of the branches of the fifth nerve or have their seat in the gas-

serian ganglion. Tumors of the pons, inflammatory and traumatic basilar lesions may produce this result.

Among the *trophic symptoms* produced by diseases of the fifth nerve are neuroparalytic keratitis, herpes zoster, facial hemiatrophy, and vasomotor symptoms. *Secretory symptoms* include dryness of the nasal mucous membrane, diminution of saliva, and alterations of taste, and dryness of the conjunctiva (both peripheral phenomena). Lesions of the gasserian ganglion are said to give rise to Horner's syndrome though this is disputed by Stewart, Oppenheim, and Villiger; Horner's syndrome is unilateral myosis, ptosis, enophthalmus, and anidrosis of the face, caused by paralysis of the cervical sympathetic, because of lesions of sympathetic fibers that pass through the ganglion to the iris

Lesions of the Seventh Nerve: *Total palsy* is characterized by unilateral facial paralysis which is recognized on the paralyzed side, by drooping of the corner of the mouth, flattening of the nasolabial fold and the frontal folds, widening of the palpebral fissure, inability of showing the teeth, of whistling, of inflating the cheek, of wrinkling the forehead, and of *closing the eye completely (Bell's palsy)*.

Supranuclear palsy (contralateral) is characterized by the relative noninvolvement of the muscles supplied by the upper facial distributions Occasionally, these muscles may move only emotionally. The corneal reflex is preserved

In nuclear and peripheral lesions the eye on the paralyzed side may appear higher than on the sound side when the eyes look upward.

When the nerve is paralyzed: (*a*) Below the stylomastoid foramen, there is more or less motor paralysis.

(*b*) Between the origin of the chorda tympani and the branch to the stapedius there is complete *motor paralysis, ageusia* in the anterior two-thirds of the tongue on the paralyzed side and diminution of submaxillary secretion of saliva.

(*c*) Lesions between the nerve to the stapedius and the geniculate ganglion result in complete motor paralysis, ageusia, diminution of the salivary secretion and hyperacusis.

(*d*) Lesions between the geniculate ganglion and the internal auditory meatus cause complete motor paralysis, no disturbance of taste, diminution of saliva and of the secretion of tears.

(*e*) In lesions at the base of the brain there is frequently added an eighth nerve lesion.

Contractures of one-half of the face frequently follow seventh nerve lesions

Facial spasm may accompany irritative central and peripheral lesions of the seventh nerve and lesions of the fifth nerve. It may be of all degrees Peripheral lesions are usually accompanied by a simultaneous spasm of *all* the muscles involved

Bilateral peripheral seventh nerve lesions occur in basilar meningitis, especially syphilitic, in aneurysm of the vertebral artery, and in bilateral middle ear disease.

In testing the sense of taste, salt, vinegar, sugar, and quinine are used. The patient protrudes his tongue, a minimum quantity of the substance to be identified is placed on the tongue and the patient points toward one of several slips that bear the terms, salty, sour, sweet and bitter, according as he experiences the respective taste sensation.

Lesions of the Eighth Nerve: These may be located in the cochlear or vestibular branches.

Lesions of the Cochlear Nerve: These are usually accompanied by deafness or hypacusis. Tumors of the brain stem, of the cerebellum, especially of the *cerebellopontine angle, may cause these* lesions. Tumors of the cerebellopontine angle also cause lesions of the fifth, sixth, seventh and vestibular nerves, and sometimes of other cranial nerves Cochlear nerve deafness is seen in cerebral syphilis. Irritative phenomena on the cochlear nerve include tinnitus, various ear noises, and hyperacusis

Lesions of the Vestibular Nerve: These are elicited by the *Bárány tests* which are performed by rotating the patient about a vertical axis, with his head in various positions (Barany chair method —the different semicircular canals are tested when various positions are assumed by the patient). By nystagmus produced by syringing the ears with hot and with cold water; by vertigo and nystagmus (or the lack of these), produced by the passage of a galvanic current of from two to four milliamperes with the electrodes on the mastoid processes during the passage (normally the head turns toward the positive pole), and by past-pointing tests before and after these procedures.

The vestibular syndrome as ascertained by the help of these tests consists of syndromes of deficiency and syndromes of irritation. The syndromes of deficiency are characterized by want of some of the normal reactions. The syndromes of irritation are found in the attacks of paroxysmal vertigo characteristic of Ménere's disease

Lesions of the Ninth Nerve: This nerve is rarely paralyzed alone; its characteristic is the palsy of the superior constrictor muscles of the pharynx which interferes with the swallowing of solid food. Sometimes taste is affected in the posterior third of the tongue.

Lesions of the Tenth Nerve: Supranuclear lesions, if unilateral, usually *give rise to little or no trouble,* because of the bilateral cortical innervation of the parts supplied. In nuclear lesions the soft palate and the vocal cord on the side of the lesion are paralyzed. Peripheral lesions resemble nuclear lesions, but may not be so generalized.

Lesions of the Eleventh Nerve: Here the sternomastoid and the upper part of the trapezius muscles are paralyzed. Supranuclear lesions, if unilateral, do not give rise to nuclear trouble, on account of bilateral innervation. Nuclear lesions are associated with the condition of the palate and the larynx paralysis described under lesions of the tenth nerve. Peripheral lesions are not apt to be so generalized as nuclear lesions, and may be seen in Pott's disease and in aneurysms of the vertebral artery.

Spasm of the sternocleidomastoid and trapezius muscles is part of the symptomatology of the condition known as "spasmodic wryneck." In this condition the centers affected are probably cortical.

Lesions of the Twelfth Nerve: Supranuclear lesions are followed by contralateral paralysis and without atrophy of the tongue and without reactions of degeneration. In nuclear and infranuclear lesions the tongue shows wasting, appears wrinkled and fibrillary tremors are present. The sense of taste is not interfered with. In pseudobulbar palsy the whole tongue is paralyzed as well as the muscles of the lips and pharynx, and possibly those of phonation, involving also other cortical or supranuclear centers or tracts. Unilat-

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DIFFERENTIAL TABLE OF SIGNS OBTAINED BY THE BÁRÁNY TESTS.

	Cerebellum	Cerebellopontine Angle Auditory Nerve	Pons	Labyrinth
Nystagmus before stimulation of vestibular nerve.	Spontaneous nystagmus may or *may not be* present. It usually is pres*ent.*	May or may not be present. *Usually* spontaneous if present.	May or may not be present. Often only present when eyeballs are moved	In acute cases of inflammation spontaneous nystagmus which gradually diminishes in severity, is present. In chronic cases it is usually absent.
Nystagmus after douching or turning.	Increased	Not increased.	May be absent or weak	Not *increased*
Past pointing	Absent or points to wrong side	Absent	Is present if the horizontal canal is stimulated by turning with head at 30° forward or by cold douching with head 90° backward or douching with head 30° forward.	Is absent or to wrong side, or the patient does not point as far past the point as he should.
Hearing	Good	Diminished or absent	Good	Diminished or absent.
Vertigo	Not [illegible] Subj[illegible] tatio[illegible] from [illegible] lesion.	[illegible] of tumor. Tinnitus aurium	[illegible] absent, may [illegible]ht	Paroxysmal attacks Tinnitus aurium
Symptoms of asynergy.	Present and well marked.	Usually present, but not so well marked as in intracerebellar tumors.	May be slight or absent	Absent

eral nuclear lesions are seen in bulbar hemorrhage and softening, and in bulbar palsy. Here there are reactions of degeneration. Peripheral lesions are seen in suboccipital Pott's disease, meningitis, tumors, fractures, bone caries, and injuries to the base of the skull.

The ninth, tenth and eleventh cranial nerves are often affected together. This may occur unilaterally in the so-called syndrome of the posterior lacerated foramen, or the *syndrome of Vernet.* The tenth, eleventh and twelfth nerves may be paralyzed together, in the so-called *syndrome of Jackson* (Hughlings Jackson), or the tenth and twelfth nerves, as in the so-called *syndrome of Tapia* (a glossolaryngeal paralysis, unilateral, sometimes due to a lesion of the trunks of the tenth and the twelfth nerves where they cross in the pharyngomaxillary triangle). Tapia's and Jackson's syndromes may be caused by central lesions as well as by peripheral lesions

Lesions of the Spinal Cord

The symptoms commonly encountered in diseases of the spinal cord depend upon the nature of the lesion, *i. e.*, syphilis, tumor, irritation, compression, hemorrhage, degeneration, etc., and upon its position and extent, *i e.*, whether the entire cord, part of it or various segments are involved. In general, the manifestations are usually below the level of the lesion; they are bilateral though at times asymmetrical, and show segmental distribution of either sensory or motor defects. There may be sensory and motor disturbances such as paraplegia, disturbance of gait, disturbance of reflexes and of sphincteric control.

Syphilis of the Spinal Cord: Syphilis has the unique distinction of being able to cause disease of any part of the nervous system Therefore, the symptoms produced by neurosyphilis are many and varied and may simulate any organic or functional disease of the nervous system. The lesions most commonly encountered are cerebral syphilis, cerebral gumma, cerebrospinal syphilis, spinal syphilis, syphilitic meningitis, and peripheral nerve affections. These may cause sensory or motor disturbances or both. Syphilis may also cause mental symptoms such as are found in general paresis and may cause psychosis and hallucinations. The various lesions may be caused by either acquired or congenital syphilis

Tumors of the Spinal Cord: The tumors may be of three types: Intradural (within the membranes); intramedullary (within the spinal cord), and extramedullary (outside the spinal cord) There are also extradural tumors which involve the vertebrae. These tumors are usually metastatic. The intramedullary tumors are more often glioma.

Symptoms presented may be due to irritation or to compression.

The Irritative Symptoms: The irritative symptoms may be sensory or motor. Pressure on the posterior roots causes either unilateral or bilateral pain at the level of the distribution of the nerves involved. There may also be hyperesthesia giving rise to the sensation of burning or to searing pain. If the irritation occurs in the cervical region, it will also affect the sympathetic fibers Pressure on the anterior roots and the anterolateral columns will cause spontaneous muscle spasm of the arms or legs The spasm may be involuntary, occurring suddenly. In the lower extremities, the thighs may be flexed upon the abdomen and the legs on the thighs If flexion of the foot occurs, the ankles and the big toe become flexed This may or may not be accompanied by pain. Occasionally this reflex may be brought out by irritating the skin

Compression Symptoms: Compression of the spinal cord may be caused by tumors, arachnoiditis, myelitis (acute or chronic), fractures and dislocations of the spinal vertebrae, tuberculosis, aneurysm, Hodgkin's disease, and parasites within the spinal canal. The symptoms depend upon the site of the compression, its extent, the accompanying spinal root involvement and the amount of interference with its vascular supply.

Complete Transverse Lesion: This will cause total flaccid paralysis of the muscles below the level of the lesion (spastic paralysis indicates that the lesion is incomplete), rapid wasting of the paralyzed muscles with loss of normal electrical reactions, and loss of sensibility from below upward to the level of the lesion, including loss of bladder and rec-

tal control, and total anesthesia to the level of the lesion.

Hemicord Lesion: When half the cord is affected, the so-called Brown-Sequard syndrome is noted. This consists of:

On the side of the lesion (homolaterally), motor disturbances such as:

(*a*) Flaccid paralysis and atrophy of the muscles whose center of innervation is at the level of the lesion.

(*b*) Loss of critical sensibility below the level of the lesion

(*c*) A zone of anesthesia just above the zone of loss of tactile sensibility

(*d*) A zone of hyperesthesia above the zone of anesthesia.

On the side of the body opposite the site of the spinal lesion (contralateral side), sensory disturbances such as.

(*a*) Loss of pain sense.

(*b*) Loss of temperature sense.

(*c*) Loss of superficial sensibility

Diseases Affecting the Posterior Columns (sensory tracts): ***Tabes Dorsalis*** (locomotor ataxia): This is a chronic progressive disease due to syphilis affecting the posterior columns of the cord (columns of Goll and Burdach) The symptoms are girdle pain, or sharp shooting pains (crisis) in various parts of the body (gastric crisis, vagal crisis, etc), impairment of touch, vibratory and position sense, absence of patellar reflex, presence of Argyll-Robertson pupil, of ataxia, of Romberg sign and of the characteristic tabetic gait. There is also hyperextension of the knees and flat feet. The Wassermann reaction and other tests for syphilis in untreated cases are usually positive.

Friedreich's Ataxia: This is a chronic slowly progressive familial disease occurring in childhood, often in siblings. It is due to degenerative changes in the columns of Clarke, the spinocerebellar tract, the lateral and posterior columns. The symptoms are ataxia, both with eyes shut and open, the gait is stumbling or "drunken"; there is difficulty in climbing stairs, and there may be clubfoot. The knee jerk and Achilles

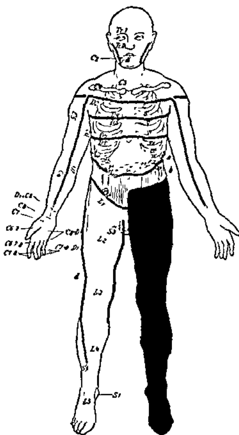


Fig. 1 — Brown-Sequard's paralysis *a*, Hyperesthesia; *b*, *c*, thermanesthesia and analgesia; *d*, motor and vasomotor paralysis and hyperesthesia in the beginning.

reflex are absent; the Babinski reflex is present The speech is slow, indistinct and slurring. Nystagmus is almost always present. The mentality may be clear or it may be retarded.

Combined Degeneration or Sclerosis of the Cord (ataxic paraplegia): This usually occurs in middle life and is progressive. The lesions are microscopic,

but generally show extensive degeneration of the posterior columns, chiefly in the middorsal region; the degenerative process often extends to the direct cerebellar and the direct and indirect pyramidal tracts of the cord, and may also involve the peripheral nerves.

Symptoms: There is at the beginning a sensation of "pins and needles," with numbness symmetrically involving the fingers of both hands and the toes of both feet; later this sensation also involves the forearms and legs. Ataxia, unsteady gait particularly at night, and astereognosis with manual clumsiness develop as the disease progresses At first there is increased knee jerk and ankle clonus with muscle spasticity. Late in the disease there is present bilateral Babinski reflex and Romberg sign Sensory phenomena are also late manifestations. They are loss of tactile, pain and thermic senses. As the disease progresses there may develop prostration and mental symptoms Accompaniments of this disease are anemia and achlorhydria. The etiology is not certain; the disease may follow chronic infections, cancer, malaria, etc, and is often found in pernicious anemia and leukemia

Diseases Affecting the Anterior Horns (motor tracts): ***Acute Anterior Poliomyelitis*** (infantile paralysis): This disease is acute in onset, usually affects children and is caused by a filtrable virus which gains entrance by way of the respiratory tract The lesion is an acute inflammation affecting the anterior horn cells of the cord and may spread to the motor nuclei of the cranial nerves and to some extent to the meninges.

Symptoms: The onset is acute with some fever and is soon followed by motor weakness, spasticity and flaccid paralysis of muscles enervated by the affected segment or part of the segment of the anterior horn. The paralysis may occur in a muscle, part of a muscle, an upper or lower extremity or it may occur in any two extremities, in the muscles of the back, the abdomen or in the diaphragm. The disease may also affect the meninges, the bulb or the cerebellum During the acute stage the spinal fluid is found to be under moderate pressure; it is clear and may contain from ten to several hundred cells. At the beginning polymorphonuclear leukocytes predominate but within a few days the predominating cells are lymphocytes. After the acute stage has passed, the affected limb shows atrophy, and flaccid paralysis. Future growth of the affected limb is inhibited and the circulation is poor.

Progressive Spinal Muscular Atrophy (chronic anterior poliomyelitis): This is a chronic progressive degenerative disease affecting the anterior horn cells of the spinal cord.

Symptoms: The onset is gradual and may first affect the small muscles of the hand, causing atrophy and clawlike deformity It then spreads to the forearm, arm and shoulder. The affected limb is atrophic, hands are limp and the scapula is very prominent. The impairment may spread to other muscles of the body, causing atrophy, flaccid paralysis and fibrillary twitchings Tendon reflexes are absent or diminished. The pyramidal tract is not affected; sensation remains intact and pain is absent. This disease manifests itself during early adulthood and is more prevalent in the male.

Amyotrophic Lateral Sclerosis: In this disease both the upper and motor neurons are affected. The lesions attack the anterior horns, the motor nuclei of the bulb and later the pyramidal tracts,

so that the manifestations are those of flaccid lower neuron paralysis associated with spastic pyramidal tract disease (Wechsler).

Symptoms: Symptomatically, amyotrophic lateral sclerosis is divided into three groups: (1) The slowly progressive form affecting the small muscles of the hands and later the arms, rarely the legs; (2) the more rapidly progressive form which begins in the shoulders and neck; (3) the bulbar form which affects the lips, tongue, palate and pharynx. Types 2 and 3 progress rapidly toward a fatal issue and type 1 may easily merge into the other types Symptoms in all three forms are flaccid paralysis in the affected muscles associated with atrophy, fibrillary twitching of the affected muscles; and hyperactive tendon reflexes of the affected parts, indicating pyramidal tract participation The abdominal reflex is retained and the Babinski reflex is absent The lower extremities are weak and show hypertonicity or spasticity. There is no pain or other sensory disturbance. Reaction of degeneration becomes manifested as the disease progresses. Speech becomes nasal and later there may be paralysis of the vocal cords Swallowing is difficult so that there is drooling of saliva Mental symptoms are usually absent, though there may be spontaneous or forced laughing or crying

Miscellaneous Diseases of the Cord: ***Syringomyelia:*** This is a slowly progressive disease probably due to a congenital neural defect. It is characterized by the formation of cavities in or around the central canal and is often associated with a gliosis. The affection usually develops in the cervical region of the spinal cord, and may affect other regions, or the entire cord; and it may reach the medulla. The tracts affected are the anterior horns of the spinal cord (motor), and the lateral columns (sympathetic and trophic); it may also affect the posterior columns (sensory), the pyramidal tracts or some of the cranial nerve nuclei.

Symptoms: Since the pathology is that of a combination of segmental, nuclear, or anterior horn disease generally associated with segmental, dissociated sensory disturbances, the symptoms are as follows: There is an early bilateral loss of pain and temperature sensation in the fingers and hands so that heat cannot be differentiated from cold, though tactile sense usually remains unimpaired. There may be a sensation of coldness, numbness and tingling of the affected part, rarely a burning pain When the anterior columns are destroyed there will be a Brown-Sequard sensory disturbance in half of the body opposite the side of the lesion associated with segmental sensory loss. When the posterior columns are destroyed there will be loss of position and vibration sense. There is also atrophy of the interosseous muscles of the hands and of other muscles The tendon reflexes of the upper extremities are abolished; the skin appears cyanotic and is cold, and there may be trophic changes in the skin and hair. Horner's syndrome, kyphosis, scoliosis, various arthropathies, and signs of pyramidal tract involvement may occur. Occasionally cervical rib may be associated with this disease.

Multiple Sclerosis (disseminated sclerosis): This is a chronic progressive disease of the central nervous system characterized by numerous and widespread patches of sclerosis of various sizes and ages throughout the white matter of the nervous system, usually sparing the peripheral nerves.

Symptoms: The disease is chronic and progressive and is characterized by many remissions and exacerbations. The onset is slow and insidious and occurs in adolescents and young adults The earliest manifestations may be weakness of one or both feet; some disturbance of sensation; temporary diplopia; nystagmus, or transient dimness of vision or central scotoma; and urinary disturbances, such as frequency, incontinence or retention. As the disease progresses there may develop motor signs, such as weakness and stiffness of the legs with spastic paraplegia. The tendon reflexes are exaggerated; Babinski reflex becomes positive (pyramidal tract involvement), and the abdominal and cremasteric reflexes disappear. The gait becomes spastic or ataxic and there is rigidity of the lower extremities The upper extremities are not as severely affected However, intention tremors in the upper extremities may be quite severe. There are also tremors of the body generally, and of the head. Speech disturbances are characteristic; they may be slowing, halting, scanning or explosive Sensory disturbances such as loss of pain, touch and temperature may become manifested when the posterior columns are affected. Loss of sphincter control is a late manifestation. Mental changes such as defective memory, lack of control and emotional disturbances occur late in the disease

Landry's Paralysis (acute ascending paralysis) · This is an acute fatal disease characterized by an ascending flaccid paralysis beginning in the legs and spreading upwards. It occurs chiefly in young adult males and may be due to a virus infection The disease is of acute onset with weakness of the legs which in a few hours develops into flaccid paralysis. The paralysis spreads rapidly so that within a few days the muscles of the trunk, chest, shoulders, arms and neck become involved, and finally bulbar paralysis sets in so that respiration, deglutition and articulation are involved All deep reflexes are lost; the sphincters are uninvolved, and sensation is but rarely disturbed. Adenopathy and splenomegaly may be present.



Fig 2—Progressive neuromuscular atrophy of familial type. (Charcot-Marie-Tooth-Hoffman type)

Familial Spastic Spinal Paralysis: This is a chronic progressive disease of childhood characterized by progressive weakness, stiffness and rigidity of the lower extremities. The gait is dragging (scissors gait) and foot drop (pes equinus) usually develops. The deep reflexes are exaggerated and there develops a positive Babinski sign and

ankle clonus. Sensation and sphincter control are unaffected.

Progressive Muscular Dystrophy (pseudohypertrophic paralysis): This condition is classified among the myopathies. Several types have been described. (1) *Pseudomuscular hypertrophy of Duchenne*, which occurs during childhood and is characterized by weakness of the legs, clumsiness, and a tendency to fall and a waddling gait. The leg muscles and later the other muscles of the lower limbs and trunk hypertrophy and subsequently atrophy.

(2) *Landouzy-Dejerine type or infantile progressive muscular atrophy of Duchenne*, which first involves the facial muscles and then spreads downward. The lips protrude, causing the tapir mouth

(3) *The Erb juvenile type* of adolescence in which the dystrophy is first noted in the shoulder girdle and then spreads to the back muscles and lastly to the thigh and arm muscles

Lesions of the Brain

Brain lesions causing pressure symptoms or causing localizing signs may be tumor, hemorrhage, abscess, aneurysm, fluid, degeneration and irritation.

Lesions of the Medulla: The manifestations noted in lesions of the medulla are varied. When both pyramidal tracts are involved, symptoms in the structures below the level of the lesion will be manifested. Occlusion of the posterior inferior cerebellar artery will cause softening in the dorso-lateral portion of the medulla with involvement of the descending root of the fifth nerve and spinothalamic tract. This causes a gross sensory paralysis, so that the face is involved on the side of the lesion and the extremities and trunk on the opposite side. This type of lesion will also show signs of involvement of the ninth and tenth cranial nerves.

Lesions of the Pons: Lesions of the pons cause paralysis on the same side along the fifth, sixth and seventh nerves and crossed paralysis in the extremities Disturbance of lateral associated movements of the eyeballs occurs often enough to be of diagnostic importance

Lesions of the Brain Stem: Symptoms found in brain stem involvement are motor or sensory and usually follow the regions supplied by the cranial nerves whose origin is in the affected part of the brain stem. Sensory and motor disturbances in the extremities and trunk are on the opposite side of the lesion, while those of the face are on the same side. Station is usually affected and there is intention tremor, nystagmus and occasionally a Horner's syndrome. This is found particularly in anterior poliomyelitis, progressive bulbar palsy, tumors, multiple sclerosis and other lesions affecting the brain stem.

Lesions of the Midbrain: This region includes the cerebral peduncles and the corpora quadrigemina (the colliculi). Lesions in the anterior part of the peduncles will cause fixed dilated pupils, ptosis and external strabismus (third nerve paralysis) on the side of the lesion and hemiplegia on the other side (*Weber's* syndrome). If the lesion involves the dorsal part of the peduncle it will cause homolateral ocular palsy and contralateral hemitremor and ataxia (Benedikt's syndrome). A lesion about the peduncle involving the infundibulum or the floor of the third ventricle may give pituitary signs or diabetes insipidus. A lesion in the hypothalamic region in the upper part of the third ventricle, blocking the

foramen of Monro, may cause flushing of *the face, head and neck, lacrimation,* salivation, hiccough and attacks of unconsciousness (autonomic epilepsy of Penfield).

Lesions of the Cerebellum: Lesions affecting the cerebellum are characterized by ataxia, incoördination when the eyes are open or shut, and weakness There is intention tremor, nystagmus, diminished muscle tonus These signs are usually on the homolateral side There is no impairment of sensation. Tumors of the cerebellum may, in addition to these symptoms, cause signs of intracranial pressure such as headache, *nausea, and choked disk.*

The cerebellar syndrome consists of Pendular knee jerks, asynergy major; asynergy minor as shown in the past-pointing test, finger-to-finger and finger-to-nose tests and five Babinski tests, incoordination of station; adiadokokinesis, rebound phenomena of Holmes, tremor of involuntary movement, irregular persistent nystagmus: cephalogyric asynergy; asynergic speech disturbance resulting *in scanning, explosive and slurring* articulation.

Cerebellopontine Angle: This region may be affected by neoplasm, inflammation and syphilis When a tumor involves the path of the eighth nerve, it causes tinnitus and vestibular signs and produces a Ménière's syndrome, which is dizziness, deafness, tinnitus occurring in paroxysms, and vomiting Other cranial nerves such as the fifth and seventh may also be implicated Involvement of the fifth nerve may cause trigeminal neuralgia with loss of sensation on the affected side. When the seventh nerve is affected, *it may produce facial hemispasm, or* twitching simulating jacksonian epilepsy Lesions in the cerebellum generally will *cause pressure symptoms.*

General symptoms of cerebellopontine angle tumors are: Tinnitus; nerve deafness; constant headache; vertigo; projectile vomiting; choked disks; spontaneous nystagmus toward the contralateral side, which is intensified by head movements;



Fig 3—Left hemiplegia. (Courtesy, M K Meyers)

ataxia and swaying toward the side of the tumor, and general weakness, hypotonia and diminished reflexes

Lesions of the Cerebrum: Lesions of the cerebrum will cause headache, drowsiness, confusion, disorientation, impairment of memory, personality changes, stupor, *hemianopsia, aphasia and occasionally* convulsions and coma. The lesions may be tumor, abscess, hemorrhage, thrombosis, or any condition that *will simulate* a space-taking lesion or cause degeneration of the brain tissue

Lesions of the Cortex: Usually monoplegias or partial hemiplegias occur, perhaps diplegia when both leg centers are involved. In cortical lesions usually only the inferior facial distribution is affected. There may or may not be anesthesia which, when present, is usually incomplete. If convulsions occur, they are apt to be of the cortical (jacksonian) type, from irritation. It must not be forgotten that jacksonian convulsions may occur in so-called idiopathic epilepsy, in uremia, in alcoholism, and in lead poisoning. Conjugate deviation of the head and the eye toward the side opposite the lesion occurs in cortical irritation localized in the foot of the second frontal convolution. Sensory irritation may give rise to peripheral pains. Sometimes there are paresthesias or anesthesia dolorosa This last is more often due to optic thalamus lesions. Contractures, hypertonia, and synkinesias are apt to occur.

Lesions of the Corticospinal Tract: Lesions of the corticospinal tract usually cause hemiparesis or hemiplegia on the contralateral side. There is little impairment of gross tactile sense, pain, temperature, and vibratory sensations unless the lesion is extensive

Lesions in the Thalamus: When the thalamic region is affected, the thalamic syndrome of Déjerine-Roussy becomes evident. This consists of contralateral hemianesthesia which is complete or almost complete for all forms of sensibility. There are exaggerated reactions to painful and thermic stimuli out of proportion to the intensity of the stimulation upon the hemianesthetic area, also contralateral astereognosis with some degree of contralateral hemiataxia, hemitremor, hemichoria and hemiathetosis, and severe spontaneous contralateral lancinating pain. There is also marked emotional disturbances as may be evidenced by unprovoked outbursts of weeping or laughing.

Cerebral Localization

Lesions of the Frontal Lobe: Lesions of the frontal lobe usually cause change in the intellectual capacity of the individual, irritability, loss of memory, disorientation for space and position, and undue jocosity. There may also be weakness of the contralateral side of the face, such as smoothing out of wrinkles, and slight lagging of an eyelid.

Lesions of the Base of the Frontal Lobe: The symptoms of basal frontal lobe lesions depend upon the area affected, so that there may be loss of sense of smell, primary optic atrophy on the homolateral side and choked disk on the contralateral side

Lesions in the Lower Part of the Left Frontal Convolution: This will cause, in right-handed persons, motor aphasia. There may also be a lack of sustained attention.

Lesions of the Corpus Callosum: Lesions of the corpus callosum are characterized by pronounced mental symptoms because of interference with the association tracts. Apraxia, difficulty in speech, and defects of memory are common. Mental symptoms often resemble senile dementia and paresis.

Lesions of the Motor Cortex: Lesions of the upper two-thirds of the motor cortex will interfere with the movements of the opposite side of the body or will cause hemiplegia. Lesions in the lowest third of the left motor cortex, in close relation to Broca's area, may cause monoplegia, affecting the arm, neck and face from above downward on the opposite side of the body,

and will also give symptoms of pyramidal tract involvement. Such lesions may be caused by thrombosis, embolism, hemorrhage or a tumor.

Irritative lesions of the motor cortex will cause jacksonian or focal epilepsy. Loss of sensation does not occur in lesions of the pre-Rolandic area.

Lesions of the Temporal Lobe: Lesions of the temporal lobe may only be recognized when neighboring structures are involved. Deep lesions in this lobe may involve the optic radiation and cause defects in the visual fields of the opposite side, frequently of sector type. When the uncinate region is affected, there may occur a peculiar epileptiform seizure characterized by an aura in which the taste and smell are involved.

Lesions of the Posterior Part of the First and Second Temporal Convolutions: Lesions on the right side in a right-handed person will produce word deafness and jargon or sensory aphasia.

Lesions of the Parietal Lobe: When the central gyrus of the parietal lobe is affected, there is loss of sense of position, point discrimination and localization, and loss of stereognostic perception, while sensations of heat, cold, touch and pain are seldom if ever affected.

Lesions of the Left Supramarginal Gyrus: These may produce apraxia and lesions in the left angular gyrus may cause alexia (word and letter blindness).

Lesions of the Occipital Lobe: Lesions in this lobe will cause homonymous hemianopsia in the contralateral fields. Irritation of the visual cortex of the optic radiations may cause visual hallucinations. Lesions of the optic thalamus are described on page 870.

Lesions in the Corpus Striatum: Lesions in the corpus striatum will produce various involuntary movements and rigidity. If the internal capsule is not involved by the lesion of the optic thalamus and corpus striatum, pyramidal tract signs will not be present on the opposite side.

Lesions in the Capsule: There is usually a period of flaccid hemiplegia or near hemiplegia which is succeeded by spastic hemiplegia with contractures. If the lesion involves the posterior part of the posterior limb of the capsule, sensation is affected. Vasomotor, secretory and trophic disturbances may occur, as well as some degree of muscular atrophy. Synkinesias may appear. Hemichorea and hemiathetosis are apt to be seen in infantile hemiplegia. Hemitremor may be seen when the lenticular nucleus is involved. Probably lesions of the accessory motor tracts are at the root of some of these disturbances of motion. Hemiataxia, increased on closure of the eyes, due to sensory disturbance, may be present

Signs indicating the paralyzed side during the stage of coma, in cerebral hemorrhage:

(*a*) Absence of the corneal reflex on the paralyzed side

(*b*) Spreading out of the thigh on the paralyzed side (breites bein).

(*c*) Raimistes' Sign: When the forearm and hand, the patient lying supine, are placed at right angles to the arm, the hands fall in flexion. On the sound side, the hand remains vertical.

(*d*) Conjugate deviation of the head and the eyes takes place to the side opposite to the paralysis.

Signs indicating the presence of a slight late hemiplegia:

(*a*) *Revilliod's Sign:* The closure of the eye on the paralyzed side is less

energetic and the eye cannot be closed alone.

(*b*) *Platysma Sign* (Babinski): There is a failure of contraction of the platysma muscle on the paralyzed side when force is opposed to the opening of the mouth, or to the downward movement of the chin.

(*c*) *Movement of Passive Supination* (Neri): If when the hand is pronated on the forearm (patient supine), the forearm is flexed by the physician, the hand tends to supinate.

(*d*) *Mendel-Bechterew's reflex.*

(*e*) *Strümpell's sign.*

(*f*) The usual signs of pyramidal tract involvement, which, however, may not be marked.

Uremia may assume a hemiplegic phase, which cannot be well distinguished from cerebral hemorrhage; it is, however, transitory. A cerebral tumor is accompanied by other signs of tumor; an endocarditis or a pulmonary abscess points to cerebral embolism. A full, slow pulse speaks for increased intracranial pressure as in hemorrhage rather than in embolism or thrombosis.

Lesions of the Anterior Part of the Internal Capsule: These produce hemiplegia on the opposite side.

Lesions in the Posterior Part of the Internal Capsule: These produce hemianopsia and loss of sensation on the opposite side.

Localization of Brain Tumors, Brain Cysts and Brain Abscesses: The general symptoms of these conditions are those due to increased intracranial pressure, *i. e.*, optic neuritis (not always present); headache; vomiting, with or without nausea, and sometimes projectile in type; vertigo; perhaps jacksonian attacks; slow pulse, and mental symptoms, such as apathy and a tendency to sleep during the day. The skull may be tender to percussion, especially in brain abscess. In the localization of the intracranial condition, a knowledge of what has been set forth under *Syndromes* is of advantage. Tumor in the *left temporal lobe* should produce sensory aphasia, and perhaps visual field limitations of the hemianopic or quadrant hemianopic type; perhaps affecting chiefly the color fields. Growths in the *occipital region* give rise to hemianopsia of the fields of vision of the opposite side; when the *inferior lip of the calcarine fissure* is involved, there is *quadrant hemianopsia for the opposite superior fields*; when the *superior lip* is involved, the quadrant hemianopsia affects the *opposite inferior fields.* Incomplete defects in the fields of vision, especially of the upper interior part, suggest a tumor or abscess in the substance of the temporal lobe. *Parietal lobe tumors* are characterized by the aphasias, with perhaps astereognosis There may be loss of deep sensibility and some ataxia. It has been asserted that in *frontal lobe tumors* mental symptoms may predominate more than in brain tumors of other parts of the brain, but this has been doubted. Pressure on the optic nerve in cases of frontal lobe tumors may occasion optic neuritis. According to Marie and Béhague there may occur in cases of frontal lobe tumor a syndrome of disorientation in space, such as inability of the patient to distinguish in the dark whether he is turning to the right or to the left. Abscess or tumor in the posterior fossa will cause choked disks, and involve the eye muscles supplied by the third, fourth and sixth cranial nerves. There will be diplopia, nystagmus, and inability of external rotation of the eye.

Tumors or abscesses in the other parts of the brain may be localized by a consideration of focal symptoms set forth in "Lesions of the Brain," p. 866.

Abscesses of the brain may occur after traumatism to the skull, after infections, in congenital heart disease (septal defects causing paradoxical emboli and abscess), and after ear or sinus disease.

Encephalography and ventriculography are valuable aids to definite localization.

Signs of Intracranial Pressure After Brain Trauma: These include unconsciousness, headache, nausea, and vomiting, local signs of head injury, shock, medullary edema (characterized by an initial slowing of the pulse and respiration, followed by a rise of these and a marked rise of temperature). The pulse rate and respiration are affected and blood pressure may rise. There may be paralyses of the extremities or of the cranial nerves, impairment of sensation, restlessness, jacksonian seizures, abnormalities of the reflexes, pupillary findings with perhaps inequality, the pupil on the side of the lesion becoming dilated when there is a cortical paralytic lesion. The increased intracranial pressure may be shown also by manometer readings of cerebrospinal fluid pressure and eyeground changes as seen by examination with the ophthalmoscope.

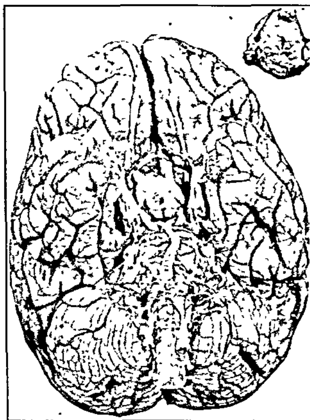


Fig 4—Pituitary tumor

Caution: In the presence of choked disks, spinal puncture should be done with caution if at all. For diagnostic purposes only, a few drops may be withdrawn.

Vascular Lesions

Meningeal Hemorrhage: Hemorrhages in the meninges are classified according to their origin. These are epidural hemorrhage, subdural hemorrhage, subarachnoid hemorrhage, and intraventricular hemorrhage.

Epidural hemorrhage usually results from traumatism The blood collects between the bone and the dura and causes the following symptoms: Headache, somnolence, and certain intracranial pressure symptoms. These may come on several hours after the injury.

Subdural hemorrhage may occur in the young, or the old, though generally in people past middle life. It may be caused by an injury, by rupture of an aneurysm or by rupture of a blood vessel. The symptoms are those of intracranial pressure which may gradually become aggravated, giving rise first to headache, visual disturbance and coma. When the bleeding is localized and forms a hematoma, it will cause pressure symptoms in keeping with the part of the brain compressed

Subarachnoid hemorrhage usually results from rupture of an aneurysm or blood vessel; the blood escaping into the subarachnoid space may trickle down around the cord. When the hemorrhage is not large, the following symptoms may be present: Some headache, retraction of the neck or nuchal rigidity, and spinal cord signs. Large hemorrhage may cause rapid death.

Intraventricular hemorrhage, when large, will cause coma from which the patient *cannot be aroused.* The face may be flushed, cyanotic and edematous; the pupils may be dilated or of normal size, *but do not react to light. In pontine hemorrhage,* the pupils are contracted. Superficial and deep reflexes are abolished, and there is flaccid paralysis. The pulse is slow and breathing is stertorous In very large hemorrhages death may occur within a short time In moderate hemorrhage, coma may last for days.

Cerebral Embolism: Cerebral embolism may occur in valvular heart disease or a portion may become detached from a thrombus. The symptoms are sudden onset of apoplexy without any prodromal symptoms. Local symptoms depend upon the site of the lesion. Consciousness may be lost at once when the middle cerebral artery is occluded. In small emboli there may be local motor manifestations with few signs of sensory disturbance.

Cerebral Thrombosis: Cerebral thrombosis usually occurs in older people. The symptoms are generally slow in onset and may affect a certain portion of the brain which would give rise to localizing symptoms. Hemiplegia is a symptom in all types of cerebral vascular disease, in hemorrhage, embolism or thrombosis. The extent and site of the lesion determine the severity of the manifestations.

Occlusion of Some Specific Brain Arteries: ***Occlusion of the Vertebral Artery:*** Here there is no picture decidedly different from that of occlusion of the posterior inferior cerebellar artery.

Occlusion of the Basilar Artery: This involves centers nearer the median line than does occlusion of the vertebral artery. The disturbances in speech and deglutition are almost or quite complete, without atrophy or reactions of degen-

eration; the disease is rapidly and distinctly progressive within a few days of the onset.

Occlusion of an Anterior Spinal Artery: It is supposed that a unilateral occlusion might lead to homolateral upper and lower extremity paralysis with sensory changes that may be on either side or may be bilateral, with failure of the tendon reflexes. Cases of this condition are rare.

Sinus Thrombosis: ***Lateral Sinus Thrombosis:*** This is usually seen in association with middle ear disease There is a sudden elevation of temperature with sudden remissions to normal or nearly normal, pronounced chills, prostration and sweats, high leukocyte count with preponderance of polymorphonuclear cells, headache, and mental symptoms such as delirium or dullness Keeler[1] mentions a peculiar mental alertness In many of the cases the symptoms are obscure An extensive lateral sinus thrombosis may extend to the jugular vein, in which case the thrombus becomes palpable.

Cavernous Sinus Thrombosis: The edema and venous stasis about the eye and in the eye are clues to the seat of the lesion. The structures in the sinus are involved, wholly or in part

Aneurysm of the Cerebral Arteries: These may affect the middle cerebral branches, the basilar, the internal carotid, the anterior cerebral, the posterior communicating, the anterior communicating, the vertebral, the posterior cerebellar, the inferior cerebellar and any of the branches forming the circle of Willis. The size of the aneurysm may vary from that of a lentil to that of a walnut or larger. These may be due to Congenital defects; endarteritis (simple or syphilitic); embolism, and periarteritis nodosum.

The *symptoms* depend upon the size of the aneurysm and its location; when large, it will cause localizing pressure symptoms. Aneurysm of the internal carotid may compress the optic nerve or the commissure causing optic neuritis, paralysis of the third nerve and hemianopia. Aneurysm of the vertebral or basilar arteries may involve the nerves from the fifth to the twelfth Aneurysm of the circle of Willis may cause hypothalamic or pituitary symptoms, such as diabetes insipidus. Rupture of an aneurysm may cause rapid death from subarachnoid or intracerebral hemorrhage

Other Organic Diseases of the Brain and Meninges

Cerebral Edema: This is not a clinical entity. It may be associated with serous meningitis, and is often found in uremia, alcoholic intoxication, and occasionally after trauma. It may also occur in tumor or abscess of the brain and in arteriosclerosis

Symptoms: These are convulsions, coma, localized paralysis, or other signs of brain compression or irritation and those of the underlying condition responsible for the edema.

Acute Hydrocephalus: This may resemble meningitis. It must be remembered that Brudzinski's and Kernig's signs may be found when meningeal irritation is present. In hydrocephalus there are dilatation of the subcutaneous veins of the head, prominence of the small fontanel which at first pulsates and then ceases to pulsate, progressive enlargement of the volume of the head and spreading of the sutures. It may be

[1] Keeler Atlantic Monthly Journal, Feb, 1926

symptomatic of brain tumor (SEE: Fig. 2, p. 159).

Encephalitis (cerebritis): *Definition:* Encephalitis is an inflammation of the brain tissue, and may be acute or chronic. It may occur as a primary disease, as a complication of local or general infections, or as a result of trauma. The brain substance alone may be affected or there may be accompanying involvement of the meninges.

Symptoms: The symptoms found in all forms of encephalitis are both general and local The general symptoms are headache, irritability, convulsions, vomiting, somnolence, delirium and coma. When the encephalitis is accompanied by a space-taking lesion, there will be signs of intracranial pressure, *i. e*, *papilledema and slow pulse. Not all of* the signs may be present at the onset. The local symptoms are those of irritation and paralysis depending upon the part of the brain affected; these may be sensory, motor or both (SEE: cerebral localization, p. 868). If meningeal inflammation coexists, there will be associated meningeal symptoms, *i. e*, nuchal rigidity, *Kernig's sign, etc The types* of encephalitis usually encountered may be lethargic (epidemic) encephalitis, traumatic encephalitis, suppurative en-

toxic infection which has a predilection for the basal *ganglia, the midbrain (especially* the substantia nigra and the oculomotor nuclei), and also for the pons and medulla, though any part of the central and peripheral nervous system may be involved The meninges are also affected



Fig. 5—Encephalitis lethargica. (Courtesy, M. K. Meyers)

but show only a moderate inflammatory reaction.

Epidemic Encephalitis (encephalitis lethargica): This is an acute, widespread and disseminated *inflammation of* the brain that may affect both sexes and all ages, though it is commoner among the young. The disease is thought to be due to a filtrable virus (not as yet definitely proven). It appears to be infectious and generally occurs in epidemics. The lesion in the brain is usually that of a

Symptoms. The symptoms of encephalitis *lethargica (epidemic)* are variable, depending largely upon two factors, namely, the severity of the infection, and the parts affected.

The symptoms usually encountered are: (1) Acute onset; (2) *fever*, 99° to 102° F.; (3) diplopia, which may precede the fever or may occur with the

temperature rise, is usually transient and may be followed by impairment of accommodation and, at times, of light reaction; (4) headache; (5) signs of meningeal irritation, such as mild nuchal rigidity, suggestive Kernig's sign, etc.; (6) drowsiness, apathy, lethargy and stupor and, at times, coma from which the patient may be momentarily aroused to carry out commands or to answer questions; (7) evidence of cranial nerve involvement (palsies); (8) abnormal involuntary movements; (9) tendon reflexes are seldom affected; (10) abdominal reflex may be hyperactive; (11) catatonic attitudes; (12) masklike immobile facies and saliva drooling from the mouth; (13) other signs and symptoms occasionally encountered are: Insomnia; delirium; various mental symptoms; myoclonic movements of various groups of muscles; radicular pain; peripheral neuritis; incontinence of urine, and, at times, retention of urine. In the bulbar type, there is respiratory difficulty, irregular pulse, inability to swallow and high fever.

The spinal fluid is under some increased pressure; it is clear and may contain from 10 to 50 or more lymphocytes The globulin and sugar content are increased.

Classification: Wechsler[1] lists eight types based on "anatomical localization" (1) The lethargic mesencephalic group, characterized in the main by stupor, pupillary abnormalities, and ocular palsies; (2) the hyperkinetic and basal ganglion group with abnormal movements, such as tics, myoclonias, choreic, athetoid, and dystonic movements, (3) the psychotic group with cerebral symptoms, such as delirium or mania, and catatonia, in addition to stupor; (4) the large basal ganglion or substantia nigra group with parkinsonian rigidity and tremor, the so-called *amyostatic* variety; (5) the meningitis group, closely simulating tuberculous meningitis; (6) the bulbar group, often fatal, with paralysis of deglutiton, respiratory and cardiac failure; (7) the neuritic group; (8) the myelitic and myeloradiculitic.

Prognosis: The bulbar type has an overwhelming mortality. Death usually occurs within several days after the onset of severe bulbar symptoms. The mortality of other types is less than 20 per cent. The greatest majority of those surviving develop definite residual manifestations Parkinson's syndrome (paralysis agitans, SEE p. 882) is the commonest sequel; it may be mild or severe Other sequelae may be various types of psychoses, neuroses, hysteria, narcolepsy, catalepsy, personality changes and myotonic manifestations

Traumatic Encephalitis: This may develop soon after a head injury or sometime later as the result of a vascular injury or abscess formation The symptoms depend upon the site of infection and the amount of brain involvement; they are usually not intense, though they may present many of the usual signs of encephalitis in a more chronic form.

Alcoholism: This may show signs of encephalitis but the signs are usually more localized

Botulism: This often closely resembles encephalitis. The attack is acute, usually afebrile; the pupils are dilated and fixed; bulbar symptoms are marked; there is great prostration and weakness, and meningeal symptoms, if any, are few. There is usually a history of having eaten spoiled ripe olives or other spoiled foods (canned meats, beans, etc.)

[1] Wechsler, I S: "Clinical Neurology," 4th Ed, p 424, W. B Saunders Co, 1940

Differential Table of the Various Types of Encephalitis and Allied Conditions

Most of the acute encephalopathies have many common gross clinical manifestations There exist however individual differences that are pathognomonic of specific types Acute encephalitis may be due to a general infection, a local lesion, or it may be postinfectional as after vaccination, measles, chickenpox, smallpox, herpes zoster, mumps, bacterial endocarditis and many other acute infections

Disease	Onset	Duration	Temperature	Pulse Rate	Respiratory Rate	Age	Season	Clinical Manifestations	Infectious Organism	Blood Findings	Spinal Fluid
Lethargic Encephalitis (Economos) (Epidemic)	Gradual or abrupt	Hours to weeks or years 25 per cent to 40 per cent fatal	Irregular May be high or low.	Follows temperature	May follow temperature Often slow	Third to fourth decade	Winter and spring	Depend upon type and stage of disease, somnolence, ophthalmoplegia, upper motor neuron reflexes, tremors.	Virus (suspected)	Leukocytosis moderate, no specific antibodies formed	Pressure, slight increase Leukocytes 20 to 200. Globulin + Glucose +.
St. Louis Type Encephalitis.	Abrupt, one to five days.	About one week 20 per cent fatal	High	Follows temperature.	May follow temperature, or slow	Any age, chiefly past 40	Summer	Vertigo, nausea, vomiting, nuchal rigidity, difficulty of speech, mental confusion Lethargy not always present Reflexes, upper motor neuron	Virus	Leukocytosis moderate Neutralizing antibodies in those recovered	Clear, pressure moderate 50 to 1000 *lymphocytes.* Globulin +. Glucose ±
Equine Encephalitis	Abrupt	Two to ten days 70 per cent fatal	103° to 105°F.	Variable	Rapid	Chiefly children	Summer or early autumn	Convulsions, coma, nuchal rigidity stiffness of back muscles. Kernig's sign positive Some edema of face and lower extremities.	Virus spread by tick or by Aedes mosquito	Leukocytosis Neutralizing specific antibodies seven to ten days of onset	Clear, increased pressure 200 to 2000 Leukocytes chiefly polys. Globulin+++ Sugar not increased
Australian X Disease	Abrupt	About one week 70 per cent fatal	High	Follows temperature	Follows temperature	Most young children	Summer	Drowsiness, nuchal rigidity, myoclonic movements, twitching increased upper neuron reflexes	Virus	Neutralization of virus with specific antibodies	Clear, about 100 lymphocytes Pressure ± Globulin +. Glucose —
Japanese Type B Encephalitis	Abrupt	One to two weeks	High	Follows temperature	Follows temperature	Older people	Summer	Drowsiness, nuchal rigidity, irritability, disorientation, at times maniacal Increased upper motor neuron reflexes	Virus	Leukocytosis. Neutralization of virus with specific antibodies	Clear, about 100 lymphocytes Pressure ± Globulin + Glucose —
Toxoplasmic Encephalitis	Gradual	One to four weeks	Moderate to high	Follows temperature	Follows temperature	More often in children	Any season	Depends upon severity of infection Headache, irritability, nuchal rigidity, convulsions, enlarged spleen and lymph nodes, jerky, clonic spasms	Toxoplasma (a protozoon)	Moderate leukocytosis high erythrocite count, neutralizing specific antibodies	Clear, 30 to 200 leukocytes Globulin normal Glucose — Toxoplasma can be isolated from guinea pigs inoculated with spinal fluid or tissue
Torula Meningoencephalitis.	Variable	Two to four weeks or longer	Moderate	Follows temperature	Follows temperature	Any	Any	Headache, anorexia, jaundice, nuchal rigidity sluggish tendon reflexes, Kernig's sign ±, general lethargy	Torula *histolytica* (fungus)	Nothing characteristic	Clear, pressure high, 20 to 60 lymphocytes protein and sugar normal Torula histolytica may be isolated from spinal fluid in culture or smear

This condition is caused by the *Bacillus botulini*

Syphilitic Encephalitis: This may occur with paresis or other syphilitic encephalopathies. It is generally chronic, and may show various general and focal signs of brain disturbance. The blood and spinal fluid will usually give a positive syphilitic reaction. Fever is generally absent, and antisyphilitic treatment almost always causes a rapid regression of symptoms.

Meningitis (disease of the meninges): The meninges enveloping the brain and spinal cord are subjected to inflammatory changes which may be acute or chronic. When the inflammation affects the dura mater, it is known as *pachymeningitis*; and when the pia and arachnoid membranes are affected, it is termed *leptomeningitis*. The entire brain covering or only a portion thereof may be affected. Occasionally, the coverings of both the brain and spinal cord may be involved. This is known as *meningomyelitis* Inflammation of both the meninges and the brain substance is known as *meningoencephalitis*

Etiology: The causes of meningitis may be trauma, tumor, hemorrhage, syphilis, or infections. The infection may be secondary to infections elsewhere in the body or it may be due to primary invasion of the meninges and cerebrospinal fluid by specific organisms, each causing a specific type of meningitis.

Signs and Symptoms common to all types of meningitis irrespective of its etiology: The four constant or cardinal signs of meningitis are

(1) *Headache:* This is the commonest and earliest symptom; it is generally diffuse, but may at times be localized in the frontal or occipital region and may extend to the nape of the neck. The pain is increased on motion and often by noise or by light.

(2) *Nuchal Rigidity:* This may be slight at first but increases as the disease progresses, later causing retraction of the head.

(3) *Fever:* This may be high at the onset or it may rise gradually.

(4) *Kernig's sign* (SEE: p. 837) is an early sign.

Brudzinski's sign (SEE: p. 841) is common in meningococcic and tuberculous meningitis.

Other signs common in meningitis are: Dizziness; vomiting; convulsions; mental or psychic manifestations, such as restlessness, irritability, apathy, drowsiness, and, at times, insomnia or stupor; coma and delirium are late signs. There may also be hyperesthesia of the skin, sensitivity to light and sound, sluggish pupillary reaction, ptosis, strabismus with diplopia and signs of aphasia or palsy The spinal fluid is under increased pressure. The presence of the four cardinal signs indicates meningitis; the type is diagnosed by the examination of the spinal fluid secured by lumbar puncture.

Purulent Meningitis: This is due to infection of the meninges by pyogenic microörganisms It is a cerebrospinal leptomeningitis resulting from trauma to the skull, otitis media, caries of the tip of the temporal bone, sinus thrombosis, sinusitis, lung abscess, infection upon the face and neck, *i. e*, carbuncle, pyemic infections, erysipelas, etc. The signs and symptoms are those of meningitis. The spinal fluid is under pressure and may be clear during the very early stages, but soon becomes turbid or purulent. The cell count is high and the predominating cells are polymorphonuclear leukocytes. The protein content is increased and the

glucose and chlorides are diminished. The causative microörganism may usually be demonstrated in the smear or culture.

Meningococcic Meningitis (cerebrospinal fever) This is an acute infectious disease often occurring in epidemic form. It is caused by one of several types of meningococcus intracellularis of Weichselbaum. The onset is acute with fever, headache and often vomiting. There may be pain in the neck and in the muscles of the limbs and joints, some degree of upper respiratory catarrh, photophobia and intolerance to noise. The posture is characteristic; the patient lies on one side with legs drawn up. The face flushes frequently, the pupils are dilated and react sluggishly; the neck is rigid. The abdomen is retracted; superficial reflexes are abolished, and Kernig's sign is positive. A macular, petechial or rose-colored spot eruption may appear over the trunk, neck or extremities during the first week of the disease The pulse is slow in comparison to the temperature. A leukocytosis of 20,000 to 30,000 with great increase in the polymorphonuclears is found early in the disease. The spinal fluid is under considerable pressure; it is turbid and contains a large number of cells in which the polymorphonuclear leukocytes predominate. During the very early stage and in the very late stage, the lymphocytes may at times predominate. The glucose content is very much lowered or may be absent; the chlorides are often reduced and the globulin is increased. The findings of meningococci in the fluid makes the diagnosis positive.

Other Types of Acute Purulent Meningitis: These may be secondary to pneumonia, typhoid fever, gonococcus infection, bacterial endocarditis or any other acute or subacute bacterial infection. The symptoms are those of meningitis and the infecting organisms may be isolated from the spinal fluid.

Acute Serous Meningitis: This type of meningitis is associated with edema of the brain (wet brain). It may occur with otitis media, whooping cough, pneumonia, measles, influenza or other infections, or it may result from a head injury and from alcoholism. The symptoms of meningitis are mild: Some headache, slight or moderate nuchal rigidity; occasional vomiting, and subfebrile or normal temperature. The pulse is slow, and there may be optic neuritis or choked disks. The spinal fluid is under pressure, but is clear and there is a moderate increase in its cellular content; the chemistry is normal and organisms are absent. Spinal drainage often relieves the condition.

Aseptic Meningitis: This may result from lumbar puncture or from the introduction of serum or lipiodol into the spinal canal. The symptoms are severe headache, some nuchal rigidity, occasional positive Kernig's sign. The temperature is usually normal.

Acute Lymphocytic Meningitis (choriomeningitis): This is a comparatively benign form of meningitis, the specific cause of which is unknown. It is believed that it is probably related to epidemic encephalitis. The onset is fairly rapid; the temperature range may be between 100° and 102° F. The meningeal symptoms are comparatively mild: Some headache; moderate rigidity of the neck, and occasionally the characteristic coiled posture. The spinal fluid is under pressure and may be clear or only slightly turbid. The cell count may vary from several hundred to over a thousand per cm., nearly all lymphocytes. The

protein is increased and the sugar and chlorides may be slightly increased.

Abscess of the Brain: This may simulate meningitis and often terminates in general meningitis. The period between the infection and the onset of symptoms may be several weeks. The temperature is low or may be normal or subnormal; the pulse is slow. Choked disks are common, and focal signs referring to various regions of the brain are usually present. The spinal fluid may be under pressure, is usually clear, has an increased cell count but no organisms. The meningeal signs are not marked.

Meningismus: The symptoms of meningismus closely simulate meningitis of a milder form. It may occur in the course of acute infectious diseases, such as influenza, pneumonia, typhoid fever, typhus fever, or other infectious fevers. At times an acute infectious disease may be ushered in with meningismus and may be mistaken for meningitis. The spinal fluid, however, shows no abnormal findings The normal spinal fluid, together with the characteristic findings of the underlying disease, is of value in differentiating meningismus from meningitis.

Acute Spinal Leptomeningitis: This rarely occurs without involvement of the meninges of the brain. It may, however, result from trauma, such as a fractured spine; it may follow spinal operation, lumbar or dorsal spinal puncture, injections of spinal nerve roots, and rarely it may result from tuberculosis of the spinal vertebra or aneurysm of a spinal artery.

The *symptoms* are severe pain in the back, pain, hyperesthesia and muscular spasm along the distribution of the affected spinal nerves The deep and superficial reflexes are at first exaggerated and later are abolished; and there may develop paralysis with anesthesia of the extremity.

Chronic Meningitis: Chronic meningitis is seldom general. Local inflammation of the meninges may be caused by syphilis, tuberculosis, tumors, embolisms, abscess and trauma. The diagnosis of underlying disease will help to determine the cause of the meningitis

Tuberculous Meningitis: This type of meningitis usually runs a subacute, somewhat chronic, course. Occasionally in children the course of the disease may be fairly rapid. The onset is slow and may not show characteristic signs of the disease for a week or more after the onset. The temperature rises gradually; rigidity of the neck becomes progressive; headache is an early symptom In children the disease may be ushered in with vomiting and convulsions. Generally during the early stages there is listlessness, with a gradual rising temperature. As the disease progresses, the signs of meningitis become increasingly more prominent. The cerebrospinal fluid is under considerable pressure. At first it is clear; later in the disease, when the fluid is allowed to stand, it will form a cloud at the top of the test tube; still later it becomes flaky and turbid The albumin content is increased. The chloride content is decreased, often below 650 mg.; and the sugar is also decreased The cell count may range from 20 to 200; the lymphocytes preponderate. The cornea may show miliary tubercles. The Magnus and deKleijn reflexes may be positive. The finding of tubercle bacilli in the spinal fluid or the positive response of a guinea pig inoculation makes the diagnosis positive

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glucose and chlorides are diminished. The causative microörganism may usually be demonstrated in the smear or culture.

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symptom that is often associated with parkinsonism is palilalia, or the repetition of short phrases in talking. Micrographia or smallness of handwriting is also seen in paralysis agitans and parkinsonism, but, as Wilson points out, may be found also in cerebral arteriosclerosis and cerebral syphilis.



Fig. 7—Torsion spasm.
(Courtesy, M. K. Meyers.)

Torsion Spasm *(dysbasia lordotica progressiva, or dystonia musculorum deformans)*: This is a disease that begins in childhood. It may be confused with Wilson's disease and with bilateral athetosis It is characterized by twisting movements of the extremities, lordosis, and spine-twisting clownish contortions. Its pathology is uncertain

Diseases of the Vegetative Nervous System

The division of the vegetative nervous system into two opposing forces for the control of the circulation, digestion and other bodily functions where one system alone might function, indicates the necessity of a very precise control of these as well as of other functions of the body. The division of the body into various systems is a recent classification by physicians to enable them to study more specifically the structures and functions of isolated parts. Actually, the body as a whole is the sum of all its component parts, and one portion of the body both influences all other parts and is equally influenced by them.

The type of disturbance depends upon whether the sympathetic division, the parasympathetic division or both divisions of the autonomic nervous system are affected. When the entire sympathetic division is chiefly affected, it may cause the syndrome known as *sympathicotonia*; if the entire parasympathetic division is chiefly affected, it may cause the syndrome known as *vagotonia*, and if the entire vegetative nervous system is affected it may cause *autonomic ataxia*, a condition in which there is evidence of dysfunction of both the sympathetic and parasympathetic divisions of the vegetative nervous system (SEE: p 825).

Angioneurosis (Tropho- and Vasomotor Neurosis)

There is a group of allied diseases which seem to have a common etiologic factor, and show evidence of some vascular and trophic disturbances The etiology is not definitely known It seems that the immediate cause is attributable to functional disturbance of the vegetative nervous system. Some of these diseases show evidence of parasympathetic disturbance; others show disturbance of the sympathetic, and still others show evidence of disturbance of both divisions. The remote cause may be endocrine disturbance, allergy, poisons, toxins, heredity or developmental inadequacy (neuropathic disposition). The conditions generally classified as being an angioneuro-

fatigue with ptosis of one or both eyelids, nasal speech, low blood pressure and often a secondary anemia. The fatigue becomes accentuated on motion, so that exertion cannot be continued for any length of time The muscles that are innervated by the bulb are among the first to become affected so that mastication and swallowing may become difficult. In severe cases paralysis of these muscles may develop Occasionally in association with the anemia, which is of the hypochromatic type, there may be a Plummer-Vinson syndrome Myasthenia gravis occurs more often in those having a thymus constitution and it has been found in association with tumor or hyperplasia of the thymus gland (SEE p. 788).

Pseudobulbar Palsy: This is distinguished from true bulbar palsy (SEE p 875) by the following characteristics: Presence of the signs of arteriosclerosis; the appearance of the disease after repeated strokes (at least two); association with spastic hemiparesis or spastic paraparesis; absence of atrophy and reactions of degeneration in the paralyzed muscles, and the presence of psychic symptoms.

Parkinson's Disease (paralysis agitans): This may be classified into two groups: (1) That occurring in advanced age which is attributed solely to senescence, and may be accompanied by arteriosclerotic changes in the globus pallidus, and (2) that occurring at any age following encephalitis lethargica (postencephalitis parkinsonism), in which the lesions in the midbrain and other parts of the brain are inflammatory. Parkinson's syndrome may also result from hemorrhage into the basal ganglia, or from syphilis or neoplasms affecting the basal ganglia

Symptoms: Parkinson's disease is easily recognized by the patient's immobile facial expression, tremors of the limbs, or of any one limb or member thereof. The tremors are moderate or fine rhythmic movements which may stop momentarily on attempted motion and during sleep. The rotary movements of the



Fig 6—Torsion spasm.
(Courtesy, M K Meyers)
Same patient as Fig 7)

hands are described as pill-rolling tremors. There may also be tremor of the jaw. The arms do not swing rhythmically when walking; they are extended and adducted; the forearms are somewhat flexed at the elbow and the hands at the wrists; the fingers are adducted and the distal phalanges are extended The gait is slowed, though at times it may be festinating; the steps are short. The head is extended and the body is bent forward. All movements are slowed. A

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sis are acroparesthesia, vasoconstrictor neurosis, vasomotor ataxia, vasodilation, Raynaud's disease, scleroderma, progressive facial hemiatrophy, angioneurotic edema, erythromelalgia, sympathicotonia, vagotonia and other less defined conditions.

Acroparesthesia: This is characterized by angiospasm of the fingers and other acral parts The patient complains of coldness, numbness of the finger tips with tingling or a crawling sensation of *other parts of the body. The condition* becomes aggravated during excitement or stress and during the night. Exposure *to cold causes blanching of the fingers*, tips of the nose and chin. This condition occurs oftener in women of the menopause age, though it may occur in young women and occasionally in men. Trophic phenomena are absent.

Vasoconstrictor Neurosis: This *condition, of which acroparesthesia may* be an expression, may produce a condition of pseudoangina It is apt to be associated *with sex difficulties.*

Vasomotor Ataxia: This includes secretory and trophic phenomena in addition to vasoconstriction.

Vasodilation: *This may occur as a* vasomotor neurosis

Raynaud's Disease: This is a severe paroxysmal, symmetrical, distal angiospasm, which during the early stages comes on in paroxysms of numbness, pallor and coldness of the fingers, toes, ear lobes, and nose. It is followed by venous stasis, that passes typically through three stages; that of local syncope, that of *asthenia*, and that of gangrene. Fingers and toes are the parts usually affected. It is commonest among women. It must be distinguished from benign vasomotor neurosis and from Buerger's disease—thromboangiitis obliterans. Mild *forms without gangrene constitute* at times the acrocyanosis chronica anesthetica of Cassirer (SEE: p. 537).

Scleroderma: This is a trophic disease of the skin and soft parts which may occur in the partial, diffuse, macular, and symmetrical forms In the severe forms there are hard edema, induration, and atrophy, with changes in the fingernails.

Morphea *is a benign form of circumscribed scleroderma.*

Facial Hemiatrophy: This is a rare condition that may be acquired or congenital. It usually occurs in young children or adolescents, though it may be acquired at any age. It is seen oftener in females and on the left side. Facial hemiatrophy may be due to a lesion in the cervical sympathetic, tumors of the gasserian ganglion, in polioencephalitis involving the nucleus of the facial nerve, and it is found in tabes and syringobulbia. In many cases an ascribable lesion is not demonstrable. The onset is gradual with mild sensory symptoms, *such as* pain and dysasthesia. Atrophy begins at the orbit, cheek, jaw and finally spreads entirely over one side of the *face, and* may involve the same side of the neck and arm. The other half remains perfectly normal. On *the affected side the* skin becomes thin and atrophic; the subcutaneous fat disappears; the muscles show signs of atrophy, and *the bones* frequently atrophy. The ear, tongue, palate and larynx show atrophic change. The hair on the affected *side either falls out* or becomes white. Horner's syndrome, when present, accentuates this unilateral facial atrophy. *The general health* is usually unaffected.

Congenital hemiatrophy has been found in several members of a family or clan No cause has been found

Angioneurotic Edema: This is a circumscribed edema of the skin or viscera, or joints, or parts such as the larynx or glottis, apparently idiopathic or, for want of an ascertained cause, regarded as functionally nervous in origin. It may be associated with urticaria and with endocrine disorders. A special variety is intermittent hydrops of the joints Rarely the functional *influence* may be purely hysterical. Similar edemas may occur in organic nervous diseases *In certain cases edema that seems to be* of angioneurotic nature may be chronic (SEE: p 927)

Erythromelalgia (*Weir Mitchell's* disease) This is a symmetrical vasomotor neurosis of the lower extremities and feet, rarely of the upper extremities, associated with slight fever, pain in the affected parts, circumscribed redness beading of the arteries and dilatation of the veins It may occur in attacks, or as a subacute or chronic affection The affected skin sweats, but trophic changes are rare

From the standpoint of the differential diagnosis of erythromelalgia the following must be excluded All acute and chronic *inflammations, e g.* erysipelas, phlegmon, gout, erythema nodosum, erythema multiforme; also the condition of erythrocyanosis symmetrica, a common and harmless vasomotor neurosis, which affects symmetrically and superficially the lower half of the legs, the forearms, and also in rare cases affects the upper arms and breasts of young girls who are otherwise chlorotic and lymphatic (SEE: p 738)

Vagotonia and Sympathicotonia (SEE p 827)

"Functional" Diseases of the Nervous System

Many of the diseases of the nervous system formerly regarded as *functional* are now, because of a better understanding of the physiology and pathology of the nervous system, included under the caption organic. Paralysis agitans is a classical example of this tendency. Perhaps most of the epilepsies and migraines are conditioned organically; and in the vertigoes, for instance, it is necessary to study the individual case so that all possible organic causes for the feeling of giddiness may be eliminated. In the absence *of any organic cause, it may be well to* look upon vertigo or tic, or headache, or migraine as functional, *a mode of response of the nervous system* (usually of a habitual nature) to certain deleterious influences, whether of environmental or of physical, physiological, or psychological nature. Most epilepsies present organic changes of the nervous system while others may be regarded as functional reactions to stimuli that seem to be endogenous and associated with changes in the chemicophysical make-up of the body fluids. It is customary to draw sharp lines of demarcation between epilepsy and hysteria or other forms of functional reaction Epilepsy and hysteria *may occur in the same individual* Epilepsy may occur as cortical or jacksonian convulsions, associated as a rule with retention of consciousness and more or less localization of the movements, as ordinary general epilepsy (grand mal); or as petit mal, in which there is momentary loss of consciousness with *little or no movement* It is distinguished from hysteria as a rule by loss of consciousness, the aura, the less showy and *noisy character of the convulsions,* the biting of the tongue and other bodily

injuries. In epilepsy, urine or feces may be passed during the attack (this may also occur in the hysterical convulsions). After the attack, a transient plantar Babinski's sign, and somnolence, and sometimes automatism may be present.

Functional reactions are hysteric, neurasthenic, psychasthenic, and anxiety neurotic, which reactions have been variously defined by various authorities on neurology and psychiatry.

Hysteria: Physically, hysteria is characterized by the expressivity of the individual affected and his ready response to suggestion. It may mimic almost any of the forms of nervous and even of mental disease, and is distinguished from these by the presence of certain associated symptoms and the absence of other symptoms that are more or less characteristic. Mentally, hysteria is characterized by a tendency to avoid conflict, and a tendency to create interest and sympathy. Hysterical psychotic states may exist in the form of "twilight states," that may last for weeks or months, seeming to reflect the fantastic excitement of dreamlike expressions and situations (Ganser's syndrome), or episodic attacks of delirium or stupor. Amnesias, fugues (actual flights from home), double personality, and even hallucinations have been noted in hysteria.

Neurasthenia: Fatigue is present on slight exertion, and may be curiously selective in that it is chiefly manifested when the patient's interest is at a low ebb. Mentally, there is inability to concentrate, uncertain memory (apparent rather than real), fear of insanity, awkwardness and self-consciousness, feelings of inferiority, irritability, phobias, and anxieties. Neurasthenia does not produce typical bodily symptoms, and the victim of it reacts inwardly, dealing with sensations, and working them over mentally.

Psychasthenia: This is marked by phobias, obsessions, marked doubts, feelings of insufficiency, nervous tension, and anxiety. Tics are often present. Marked depression and anxiety prevail.

Anxiety Neurosis: Marked anxiety or fear is the most prominent feature. With anxious expectations or dread are associated general nervous irritability and physical symptoms that may be regarded as the bodily accompaniments of fear. The intensity of the symptoms may vary. Acute exacerbations constitute the "anxiety attacks."

Mental Disease

Nomenclature and Symptomatology

In studying a patient from the standpoint of psychiatry in order to determine as far as possible the contents of that patient's consciousness, the behavior and the expressions in speech (also a form of behavior) should be observed.

In spite of the fact that it is difficult or impossible to define consciousness, Head makes the following attempt: "Consciousness is a form of integrative vital reaction, which enables the organisms to adapt themselves more perfectly to certain situations, conditioned by its internal state and the impressions produced upon it by external forces."

Consciousness varies from time to time. The unvarying nucleus of its content is known as *self-consciousness.*

Personality is a term applied to an individual's unique and practically habitual way of reacting to situations as determined by heredity and previous experience and education. According to Hun, it is the "energy of cortical activity accumulated during the life of the individ-

ual." It would be better to regard it as an aspect or tendency or way of action of energy, rather than as energy itself. In certain diseases, personality tends to split or dissociate, the individual acting with a part of his personality at one time and with another at another time This splitting may be associated with amnesia for the other personality. Such splitting occurs in schizophrenia (dementia precox), epilepsy, and hysteria

Limitations of consciousness may occur, as in epilepsy, where there may be a retraction or dimming like that of marked drowsiness or of dream states

A *percept* is the identification of an object as an object brought about by the coexistence of sensations or ideas associated with the presence of the object. Mistakes as to the identity of objects are *illusions*. *Hallucinations* are fallacies as to the actual objective existence and presence of objects, when no objects that might reasonably be mistaken for them are present

Memory is the recollection of past events The loss of recollection of events is due to a lack of registration of the events, rather than to loss of memory Loss of memory is a well-marked symptom of advancing years, it is seen in senile and presenile psychoses, Korsakoff's psychosis, and general paralysis of the insane It may be associated with falsification of memory and fabrications

The association of ideas may, in the psychoses, deviate from the normal, sound or "Klang" associations, thus taking the place of the ordinary logical associations. The direction of the flow of ideas toward a logical conclusion may be seriously interfered with.

Complexes are groups of ideas that are associated with marked affective or emotional phenomena, expressed or primarily unexpressed (repressed or suppressed). In another sense, the term complex may be applied to groups or constellations of ideas, irrespective of their associations with affective phenomena. In certain mental diseases the power of abstraction and the power of forming a logical conclusion from given premises are interfered with.

Delusions are faulty beliefs from which the mentally afflicted suffer. Perversion of the power of conclusion and abstraction may be at the basis of some of the delusions. Other factors in the formation of delusions are hallucinations, emotional states, and defects of normal voluntary response.

Emotional states may be defective quantitatively or qualitatively and may be associated with excess or deficiency of psychomotor or ordinary motor activity.

The *intelligence* suffers in dementia and feeble-mindedness. For its determination, especially in these conditions, Binet and Simon devised certain tests applicable to them. Each group of tests is responded to normally by children of definite ages. Mentally defective children respond only to tests of lower grade Various modifications of these tests, including the Terman and the Stanford modifications of the Binet-Simon tests, the Kuhlmann and other tests are used in the United States.

Classification of Mental Diseases

The following modified classification is in part from Kraepelin and in part from Strecker and Ebaugh

Psychoses Due to External Factors: I. ***Head Injury:*** (*a*) *Traumatic delirium*, disorientation, loss of memory for a period of time preceding the accident, falsification of memory, delirious

injuries. In epilepsy, urine or feces may be passed during the attack (this may also occur in the hysterical convulsions). After the attack, a transient plantar Babinski's sign, and somnolence, and *sometimes* automatism may be present.

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IV. ***Brain Syphilis:*** (*a*) Syphilitic neurasthenia; (*b*) syphilitic pseudo-paresis; (*c*) apoplectic brain syphilis; (*d*) syphilitic epilepsy; (*e*) paranoid conditions (luetic); (*f*) psychoses in tabes; these may represent a general paretic element; (*g*) the mental derangements of congenital syphilis.

Of these (*b*) may be scarcely distinguishable from genuine paresis except by laboratory tests and the absence of the classic course. During it Korsakoff's psychosis attacks may occur, and may be recognized as a special variety of brain syphilis.

(*h*) *General Paralysis of the Insane:* This may occur in demented, depressive, expansive, agitated, galloping, juvenile, and atypical forms, and forms that are associated with tabes. Early in the disease there occur changes in disposition and character, defects in judgment, unreliability, moral laxity, extravagance, forgetfulness Usually at the height of the disease and invariably in the final stages, deep dementia develops. The neurological signs (Argyll-Robertson pupils, or unequal or irregular pupils, exaggerated or absent knee jerks, tremors, speech and writing defects, convulsions), tend to make the diagnosis Kraepelin distinguishes a syphilitic pseudoparesis. The characteristic colloidal gold curve in true paresis would be diagnostic, although this may have been modified by treatment A classic course may indicate true paresis.

Psychoses Due to Internal Factors: I. ***Endocrine gland psychoses*** are due directly to hyperfunction, hypofunction, or other pathologic conditions of the various endocrine glands, seen in myxedema, exophthalmic goiter menopausal changes, hypoglycemic states, certain types of virilism, etc.

II. ***Psychoses due to endogenous brain disease*** include brain tumor, lobar sclerosis.

III. ***Psychoses due to developmental defects of the nervous system*** are Huntington's chorea, amaurotic family idiocy, tuberous sclerosis, Wilson's disease, pseudosclerosis.

IV. ***Arteriosclerotic psychoses,*** including apoplectic dementia, are often difficult to differentiate from senile psychoses. The diagnosis is justified when mental deterioration exists with evidence of general brain damage (headache, dizziness, fainting attacks), and more particularly evidence of focal brain damage.

V. ***Presenile psychoses*** exist as pernicious, late catatonic, and paranoid forms. Alzheimer's disease, an early senile deterioration, usually with rapidly oncoming dementia and with definite pathology is classed in this group by American authors.

VI. ***Senile psychoses*** include simple demented, delirious and confused, depressed and agitated, and paranoid types

In the *presbyophrenic types* there are marked memory and retention defects with complete disorientation The patient is mentally alert, attentive, and able to grasp immediate impressions. Forgetfulness leads to absurd contradictions and repetitions Suggestibility and fabrication are prominent.

VII ***Schizophrenia or Dementia Precox:*** This presents four types · (*a*) *Simple form·* Interest at a low ebb, apathy and strange behavior, delusions and hallucinations either abortive or entirely absent are characteristic. (*b*) *Hebephrenic forms:* Silliness, unexplained smiling, laughter, grimacing, manner-

perceptions, irritability, unrest. (*b*) *Traumatic epilepsy*, general or local convulsions. May be associated with mental enfeebling. (*c*) *Traumatic mental enfeeblement*. (*d*) *Traumatic Constitution:* The so-called "head syndrome" in which there is headache, disinclination to work, and emotional instability.

II. ***Intoxication:*** (*a*) *Metabolic:* Uremic, eclamptic, cancerous, cardiac, effects of thirst, heat stroke, diabetic, gouty, poisoning by phosphorus.

(*b*) *External Poisons:* Atropine, hyoscine, santonin, carbon monoxide, illuminating gas, morphine, cocaine, alcohol, etc.

Morphinism: This is associated with subjective ease of mental operation, with pleasurable sensation and lack of determination. Withdrawal is followed by characteristic withdrawal symptoms (*i. e.*, unrest, anxiety, heart palpitations, yawning, shivering, trembling, muscle spasms, sweats, diarrheas, vomiting).

Cocainism. This is characterized by activity, with hallucinations of the senses including that of the presence of insects in or under the skin, and ideas of persecution; twitchings, palpitation, sweats, insomnia.

Alcoholism: This may be acute, subacute or chronic; the toxic manifestations may be slight or severe and may present the following:

(*a*) *Delirium Tremens:* A delirium with tremor, toxic symptoms, and a prominent hallucinatory content, especially that concerned with more or less terrifying animals.

(*b*) *Acute Hallucinosis:* Hallucinosis, initially and usually predominantly auditory, with a clear sensorium, marked fears, and more or less systematized persecutory trends.

(*c*) *Korsakoff's Psychosis:* There are delirious and nondelirious types. The former are not unlike delirium tremens, although the symptoms are usually less severe and the course is longer. In the latter types, there are retention defects, disorientation, fabrication and memory falsification, suggestibility, and a tendency to misidentification of persons. There may or may not be polyneuritis. Korsakoff pictures may occur in malaria and other diseases.

(*d*) *Other types* that are not definitely classifiable, showing various stages and symptoms of intoxication, may be acute or chronic.

III. ***Infections:*** (*a*) *Meningitis*, encephalitis (SEE: pp. 874 and 877).

(*b*) *Febrile and Infectious Deliria:* Malaise, irritability, unrest, insomnia, with anxiety dreams. In severe forms there may be dreamlike states with hallucinations, anxiety, or gaiety. In severe states, amnesia with confusion and excitement. In the severest states, stupidity, lethargy, weakness and insecurity of the movements, picking at the bedclothes, deep insensibility. Postfebrile deliria are not different in kind from the febrile. There may be depression, irritability and suspiciousness.

(*c*) *Exhaustion delirium* is practically the same as (*b*) in manifestations and occurs after hemorrhage, severe overexertion, prolonged insomnia. (*b*) and (*c*) may take the form of (*d*) and (*e*).

(*d*) *Acute Confusion:* This may occur also in other types of mental diseases, such as dementia precox or manic depressive psychosis.

(*e*) *Infectious Mental Enfeebling:* This may occur after the infectious fevers, heart failure or after chronic infections.

tions of others and self deprecation. This may be accompanied by precocious senility, insomnia, disturbed nutrition, anorexia, constipation and other digestive disturbances. There may also be circulatory and pelvic symptoms The disease runs a protracted course. Shock treatment is beneficial, as is also the administration of gonadal hormones.

IX. *Senile Psychosis:* One type usually develops slowly. At first, there may be irritability of temper, insomnia, malaise, muscle weakness, anorexia, and a tendency to seclusiveness. Later, there is impairment of memory, especially for recent events. The emotions are deteriorated and there may be lack of sympathy, obstinacy, stubbornness, selfishness, self centering of interest, outbreak of temper, moral laxity and troublesome behavior.

Constitutional Psychoses: I. *Manic-depressive Psychosis:* This may be of four types· (*a*) *Mania*, predominantly excited; (*b*) *melancholia*, predominantly depressed; (*c*) *mixed* forms; (*d*) *cyclothymic basic* forms. Here the habitual emotional level of the individual is either raised or depressed Fluctuations may occur on this habitual or average level.

II. *Paranoia and Paranoid States:* True paranoia is rare. Here there is a slowly developing and logical system of persecutory and sometimes grandiose delusions, accompanied by adequate emotional response and clear and coherent thought, without hallucinations

III *Hysterical Insanity:* For hysterical convulsions, see p. 100; for hysteria, see p. 886.

IV *The various impulsive insanities* (kleptomania, pyromania, etc.):

V *The various mental aberrations* associated with sexual delinquencies.

VI. *The mental defects* associated with congenital psychopathy.

VII. *The mental states of the congenital feeble-minded.* These states are mentioned but should not be regarded as genuine insanities. A congenital feeble-minded individual, however, is not protected against the development of a true insanity.

Forms of Feeble-mindedness: The various forms of feeble-mindedness may be classified as follows:

(*a*) Forms due to meningitis, encephalitis, softenings of the brain due to vascular diseases, tuberous sclerosis, Wilson's disease, pseudosclerosis, cysts, hydrocephalus.

(*b*) Amaurotic family idiocy. A child who is born apparently normal, a few months after birth develops inability to hold up the head, has poor vision, and often has a cherry-red spot on the macula.

(*c*) Cretinism.

(*d*) Infantilism.

(*e*) Mongolism: This may be recognized by the characteristic eyes, thickened lips and tongue, drooling of saliva and low mentality.

Microcephalic and macrocephalic forms of feeble-mindedness are recognized by some authors as idiots.

Forms of Dementia: Dementias are associated with the following conditions:

(*a*) The dementias of dementia precox (Schizophrenia).

(*b*) Epileptic dementia.

(*c*) Dementia of the other forms of brain lues

(*d*) Dementias of general paresis.

(*e*) Arteriosclerotic dementia

(*f*) Senile and presenile dementia.

(*g*) The dementia of Huntington's disease.

(*h*) Dementia following head injuries

isms, peculiar and changeable ideas which have an absurd and grotesque content are in evidence. Ideas of grandeur or of persecution may occur. (*c*) *Catatonic form:* Negativism and conduct peculiarity, with phases of stupor or excitement marked by impulsive, queer, stereotyped behavior, and hallucinations are found. (*d*) *Paranoid form:* Hallucinations, delusions, particularly of persecution or of grandeur, often fairly well systematized, occur.

Dementia precox is characterized by discrepancies between thought, behavior, and emotional reaction; by emotional blunting and indifference; by seclusive make-up, silliness, defects of judgment, hypochondriacal notions, suspiciousness and ideas of reference; odd, negativistic conduct and dreamlike ideas, autistic thinking (castles in the air), and the like. The manifestations of the various types of dementia precox are often combined in the same individual.

Kraepelin separates from dementia precox a group which he calls *paraphrenia*. The individuals in this group preserve their personality intact until the end, but there are active hallucinations and delusions without the silly behavior of the precox cases The disease is progressive. It is sometimes grouped under paranoid states.

VIII ***Epileptic Insanity:*** The psychotic reactions in epilepsy are listed by Strecker and Ebaugh[1] as: (*a*) Periodical ill humor; (*b*) epileptic dreams or twilight states in which there is considerable confusion; (*c*) delirious confusion with hallucinations and ecstatic delusions or anxiety; (*d*) a "conscious delirium" in which the confusion is slight.

Epileptic furor, when it follows a seizure, is extremely dangerous. The patient is maniacal, homicidal and destructive. He may commit horrible crimes, even killing or maiming those who are near him or dear to him.

There may also be transitory states of depression and excitement, or there may be paranoic states or dementia precox.

VIII. ***Involutional Melancholia:*** This is described by Strecker as probably being a mixed form of manic-depressive psychosis in which the motor retardation is often replaced by restlessness and agitation, occurring at the climacteric in a person who had previously not shown any manic-depressive episodes. This condition is commoner among women, usually occurring between the ages of 40 and 45. In men, it may occur between the ages of 50 and 65.

Symptomatology The general behavior is variable among different patients and often from time to time in the same patient. The mood is depressed and apprehensive and there may be frenzied, agitated excitement or just restlessness There may be massive delusional formation, apprehensive and self deprecatory, concerning self, family and friends The consciousness is usually clear and orientation may be good, but the sensorium may be clouded. The patient may realize that her symptoms are abnormal or that other patients have delusions. Suicidal attempts are common. Some patients may have catatonic phenomena, such as fixed attitudes, catalepsy, negativism, stereotypy, grimacing, mannerisms, autonomic movements, food refusal, impulsive violence, restiveness, destructiveness, episodes of violent scolding, unapproachability, mutism, and retention of urine and feces. There is often repetitive speech, delusions, self accusation, accusa-

[1] Strecker, E. A. and Ebaugh, F. G. Practical Clinical Psychiatry, Blakiston Co, Phila., 4th Ed., p 187, 1940.

tometers. Severe grades of vitamin A deficiency will cause metaplasia of the conjunctival and corneal epithelium and xerophthalmia, resulting in complete blindness.

Teeth: These may become soft due to scarcity of enamel. Tooth deformities in the young and pyorrhea in adults have been attributed to vitamin A deficiency. It is quite likely that such defects may be due to general malnutrition rather than to any specific deficiency.

Respiratory Tract: Keratinization of the epithelium of the respiratory mucosa is likely to lead to bronchitis, peribronchial inflammation, bronchiectasis and severe types of pneumonia

Skin: This becomes dry and rough and may develop a papular eruption; the sweat glands may atrophy.

Gastrointestinal Tract: This may show evidence of hyperkeratosis, especially of the esophageal mucosa

The Liver: Disease of the liver fails to convert provitamins, and to store vitamin A Therefore, in the various types of cirrhosis, in other severe liver diseases and in catarrhal jaundice, avitaminosis A often develops

The Blood: It was pointed out by Abbott and Ahman that in vitamin A deficiency there is a decrease of polymorphonuclear neutrophils, a relative increase in large lymphocytes and the presence of degenerate cells.

Genitourinary Tract: There have been reports of cases of complete obstruction of the ureters and renal pelvis due to accumulation of keratinized cells. The formation of renal calculi was attributed to vitamin A deficiency. This requires further study

Nervous System: The formation of various lesions in the nervous system was noted by many observers when they fed experimental animals on a diet lacking in vitamin A.

Growth and general development may be retarded when vitamin A is withheld from the diet over a prolonged period. Vitamin A seems to be an antagonist to thyroxin and should be beneficial in hyperthyroidism. It also is said to be beneficial in senile vaginitis.

Résumé: Vitamin A is necessary for maintaining the epithelial tissue in a proper state of nutrition, thus preventing degenerative changes in the eyes, the nervous system and the various epithelial structures, and for limiting the susceptibility to infection (For availability and therapeutic use, see pp. 914, 916).

Vitamin B (Vitamin B Complex)

Vitamin B is a complex vitamin made up of an apparently heterogeneous group of specific substances, each having its own chemical and physiological properties There is, however, sufficient homogeneity in these substances to merit their inclusion into one complex group. One of their common properties is that they are all water soluble; and before they were individually identified they were classified as a single water soluble vitamin in contradistinction to vitamin A, which was known as a fat soluble vitamin.

Source: The vitamin B group (vitamin B complex) is found in most of the natural foods in sufficient quantities for the normal individual's needs The germ and the bran of cereals, as in wheat, oats, etc., and not the kernel, contain this vitamin complex. Brewers yeast is particularly rich in vitamin B complex.

The Specific Factors of the Vitamin B Group: Vitamin B_1 is synthesized as thiamin chloride, an antineuritic or antiberiberi factor.

Daily Requirements of Vitamin A: It is estimated that the minimum daily adult requirement of vitamin A is approximately from 10 to 15 U.S.P. units per pound of body weight. The average intake should be about 3000 to 8000 U.S P. units daily. The ordinary balanced diet usually contains at least that much. Pregnant women require about 6000 to 10,000 U.S P. units daily. Growing children require between 6000 and 10,000 U.S.P units of vitamin A daily.

The daily requirement of all vitamins is increased during: (*a*) Increased metabolic activity; (*b*) increased Caloric intake of food; (*c*) increased carbohydrate intake; (*d*) fevers; (*e*) pregnancy; (*f*) hyperthyroidism, and (*g*) growth.

The average vitamin A content of the commoner foods expressed in U.S P. or international units is as follows: Milk, 2000 units per pint; butter, 2000 units per ounce, one egg yolk, 600 units; cod-liver oil, 2000 to 1300 units per teaspoonful (drachm); halibut-liver oil, 600 to 7200 units per large drop The previtamin A (carotene) content of foods is, for example: Carrots, ¼ lb. fresh or boiled, 2000 units; cabbage, ¼ lb. fresh or boiled, 1000 units. Some of the green leafy vegetables contain from 1000 to 10,000 units per ounce.

The Unit: The *U.S.P.* or *international unit* of vitamin A is equivalent to 0 6 microgram of carotene and to 0 3 microgram of vitamin A.* The *Sherman unit* is expressed in "rat growth units." It represents the daily amount of vitamin A necessary to add to a standard diet (free of vitamin A) in order to have a test rat gain an average of three grams per week over a period of from four to eight weeks From 8 to 12 rats are tested so as to determine the average gain in weight. The U.S.P. unit and the international unit are identical and are more accurate than the Sherman unit. One or two Sherman units roughly approximate a U.S.P. unit.

Physiologic Action of Vitamin A: Vitamin A acts upon epithelial and other structures of the eye, upon the epithelial structures of the skin, and it, together with other factors, influences growth and increases body resistance to infection. A deficiency of vitamin A will cause degenerative changes in the various structures. An increase of vitamin A above the normal is not associated with any disease.

A deficiency of vitamin A in the body may be due to a diminished intake or to the inability of the body to utilize or to store the vitamin or the previtamin factor (carotene). This may result from disease of the liver, from coating of the intestinal mucosa by oils or by excessive mucus; and may also occur in diabetes mellitus.

Pathology: Hypovitaminosis of A affects the eyes, the teeth, the respiratory system, the skin, the digestive tract, the genitourinary tract, and the nervous system.

The Eyes: Among the early symptoms of vitamin A deficiency is night blindness or delayed adaptability to light changes caused by a deficiency of the visual purple. This fact is utilized as a test for determining the deficiency of vitamin A in the body. Various types of apparatus are now in use for determining the degree of night blindness and the rapidity with which the eyes become adapted to darkness after having been exposed to light. Two types of apparatus generally used are the Birch-Hirschfeld photometer and the Hecht and Feldman adap-

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* Booker, L. E.: "The Vitamins," A M A., p. 118, 1939.

pregnant or lactating. Many of the authors who have studied this problem fail to agree as to the exact number of units required daily. The disagreement is chiefly due to the different requirements of the various races because of differences in food habits, differences in stature of the individuals and the unequal standardization of the unit.

Cowgill[1] states that *a man* weighing about 99 pounds or 45 kilograms requires about 135 international units of the vitamin; one weighing 154 pounds or 70 kilograms needs approximately 280 international units, and still heavier persons weighing about 198 pounds or 90 kilograms require about 550 international units. The number of units, therefore, depends largely upon the number of Calories required for maintenance of the individual. Rose[2] estimated that the daily intake is approximately 15 international units or 30 Sherman-Chase units per 100 Calories of food ingested.

During pregnancy and *lactation* there is greater demand for vitamin B_1 and the intake, therefore, should be about 50 per cent more than under other circumstances

During childhood, because of growth and development, the unit intake should be proportionately greater than in the adult.

In *increased metabolic activity*, as in fevers and in hyperthyroidism, the vitamin B_1 requirement is increased

Since vitamin B_1 is easily excreted by the kidneys and the gastrointestinal tract, it is obvious that when there is polyuria or diarrhea or severe vomiting the unit intake of vitamin B_1 should be increased in proportion to the excessive amount lost from the body through these channels. Cowgill, Rosenberg and Rogoff[1] have shown by experiments on dogs that vigorous diuresis has resulted in the appearance of anorexia and of other signs of vitamin B_1 deficiency, chiefly because of the great loss of this vitamin through the kidneys.

Test for Detection of Vitamin B in the Body: Accurate tests have as yet not been devised, but an approximate idea as to the amount of vitamin B_1 in the body may be obtained by the examination of urine It has been found that normal adults excrete an average of about 12 international units daily and that excretion of less than three units is found in cases of beriberi or other types of polyneuritis It has been suggested as a test that when 350 units of vitamin B_1 are administered to a normal adult, there should be an excretion in the urine of about 30 units of thiamin chloride.

Another test utilized is the determination of the bisulfite binding power of the blood. The bisulfite binding power of the blood is expressed by milligrams of pyruvic acid. This normally ranges from 3 5 milligrams to 6 milligrams per 100 cc of blood. Elevated values indicate vitamin B_1 deficiency.

Physiology of Vitamin B_1: Vitamin B_1 has definitely proven to be an antiberiberi vitamin. Deficiency of this substance causes beriberi, certain types of neuritis and other signs of avitaminosis B_1, though not as marked as is beriberi. Vitamin B_1 also exerts a definite influence upon various metabolic processes, particularly upon carbohydrate metabolism. A lack of this vita-

[1] Cowgill, Geo. R , Ph D. Human requirements for B1—The Vitamins, A. M. A , 1939, p 236

[2] Rose, M S · The Foundations of Nutrition, 1933, Macmillan, N Y.

[1] Cowgill, G. R., Rosenberg, H. A., and Rogoff J Am J Physiol , 95: 537, Dec., 1930.

Vitamin B_2 or G is known as riboflavin which prevents or cures *cheilitis* and certain occular changes.

Nicotinic acid (the P.P. factor), which may be another factor of B_2 or is closely allied to it, is the pellagra preventative or pellagra curative factor.

Vitamin B_3 is a growth factor in pigeons.

Vitamin B_4 is an antiparalytic factor in rats and chicks.

Vitamin B_5 is a weight-maintaining factor for pigeons.

Vitamin B_6 is an antidermatitis factor for rats, and appears to have similar properties to vitamin H and factors I *and Y. B_6 seems to be of benefit in the* treatment of certain types of neuromuscular dystrophies.

Factor W is a growth essential for rats

Pantothenic acid or filtrate factor is a nutritional dermatosis preventative in chicks.

The specific factors of the vitamin B group that have thus far proven to be of clinical importance to man are vitamin *B_1 or thiamin chloride, riboflavin, nicotinic* acid (P.P. factors) and vitamin B_6

Vitamin B_1 (thiamin hydrochloride) · *Vitamin B_1 is derived from food and is also synthetized* as thiamin hydrochloride. For clinical use, the natural product is derived from yeast and other substances It is dispensed in various preparations and combinations, and under a host of trade names. The synthetic product has a definite formula and is marketed as thiamin hydrochloride, or in combination with other substances.

Food Sources: The quantity of vitamin B_1 in any one type of food is not great; in order to get an adequate *amount of this vitamin,* a variety of food is necessary. Vitamin B_1 is not stored in adequate quantities in the tissues. It is therefore necessary to obtain a daily supply of it from vegetables, such as potatoes, the legumens (raw or canned), from *fruits, nuts, whole* grains, and cereals (*not refined* flours or refined cereals); from animal foods as milk, eggs, muscle meats and organs (spleen, pancreas, kidneys, lung, liver, *etc.*). Chicken and pork are said to contain a greater quantity of *vitamin B_1 than do other meats, or milk.* Prolonged boiling or the addition of an alkali while boiling destroys the vitamin *In cooking vegetables, more of the vitamins remain in the water than in the* vegetables. Yeast is the *richest source* for all the B vitamins.

The Vitamin B_1 Unit: The unit of *Vitamin B_1* is based on the minimum quantity required to prevent beriberi in *test animals.* The two kinds of "*unit*" generally employed are the international or U.S.P and the Sherman unit.

The *international* or *U.S.P.* unit adopted at the International Vitamin Conference and recommended to the Vitamin Advisory Committee of *U. S.* Pharmacopeia *in 1938 is: "The potency* of three micrograms of thiamin chloride equals one unit of vitamin B_1." That is, *one international unit* of vitamin B_1 equals three micrograms, or three one-thousandths of a milligram of thiamin chloride. *One milligram (1 mg.) of thia*min chloride represents 333 U.S P units.

The *Sherman-Chase* unit: Approximately two Sherman-Chase units *equal* one international unit.

Human Daily Requirements for Vitamin B_1: The daily requirement of vitamin B_1 seems to depend upon the Caloric intake of food, particularly of carbohydrates, the weight of the individual, the condition of the kidney and of the bowel excretions, the age of the individual, the metabolic rate, and whether

toms are referable chiefly to the nervous system; (2) wet beriberi in which the outstanding symptoms are generalized edema or anasarca, and (3) the acute or pernicious type in which there occur serious heart symptoms that may cause sudden death. The onset of the disease is insidious with general malaise, weakness, mild gastrointestinal disturbances, diminished exercise tolerance, heaviness in the legs and cardiac palpitation. Paresthesias, soreness of the muscles and extreme sensitiveness of the nerve trunks soon follow the prodromal symptoms; this is followed by loss of superficial and deep reflexes With the occurrence of the diminished reflexes there develop edema of the legs and symptoms of cardiac decompensation The edema may vary from mild pretibial pitting to very severe swelling. At this time effusions may appear in the pericardium, the pleura and occasionally in the peritoneum The symptoms referable to the nervous system are progressive as is evidenced by the development of the steppage gait or marked ataxia, loss of tendon reflexes and electrical reactions of degeneration. There may develop at this time wrist drop and foot or toe drop associated with considerable pain Occasionally aphonia, due to vocal cord paralysis, may develop There are also mental symptoms such as confusion and occasionally Korsakoff's syndrome.

Polyneuritis Polyneuritis may occur from conditions other than vitamin B_1 deficiency. Whether polyneuritis is due to vitamin B_1 deficiency or to other causes it is benefited by the administration of vitamin B_1. When polyneuritis is due to vitamin B_1 deficiency, the onset is usually insidious, though it may be rapid, with heaviness in the legs and tenderness of the calf muscles when squeezed. Walking becomes difficult, particularly because of weakness in the legs, and if walking is persisted in after the feeling of weakness has come on, there may be sudden collapse because of the failure of the lower extremities to uphold. In milder cases there may only be burning of the soles of the feet with numbness of the dorsum and lower part of the ankle. The weakness in the extremities eventually spreads to all parts so that it affects both the extensor and flexor muscles and foot drop results The hyperesthesia is almost bandlike and is followed by anesthesia with atrophy of the muscles and of the skin over the affected part. The upper extremities usually become involved quite late in the disease although occasionally the symptoms in the upper extremities may precede those in the lower. The symptoms in the upper extremities are weakness in the hands, hyperesthesia and anesthesia with loss of tendon reflexes and often wrist drop The sphincter reflexes are usually maintained until quite late in the disease. Mental symptoms may be those of euphoria or depression The rapidity with which the symptoms spread and the length of time they may continue depend entirely upon the amount of deficiency and the ability of the individual to respond to adequate dosages of vitamin B_1 (For availability and therapeutic use, see p. 915).

Vitamin B_2 and G, Riboflavin: Riboflavin, lactoflavin, vitamin G or vitamin B_2 is a yellowish-green fluorescent, water soluble pigment found in fairly large quantities in milk, liver, kidneys, muscle, yeast, egg white and egg yolk, barley, malt, dandelion blossoms, grasses and other plants It seems to be formed primarily in the green leaves of actively growing plants, where it is found

min in the system produces a deficiency of oxygen in the heart muscle, kidneys and brain. This results from an insufficient uptake of oxygen in the presence of dextrose and an increase in pyruvic acid in the presence of lactic acid. Both of these conditions are correctible by the administration of vitamin B_1 (thiamin hydrochloride) because it brings about oxidation of pyruvic acid (Cantarow and Trumper).

Vitamin B_1 deficiency may arise from an insufficient intake, a too rapid excretion by bowel or kidneys, or by decreased absorption. Since vitamin B_1 is lost through the kidneys and the feces, it is obvious that, under certain circumstances such as diarrhea, and polyuria, an excessive amount of the substance may filter through the intestinal canal and the kidneys, thereby causing a deficiency. *In diseases of the mucous membrane* of the intestinal canal hypovitaminosis B_1 results from the deficient absorptive power of the bowel because of insufficient permeability of the mucosa.

Pathology: Hypovitaminosis of B_1 affects various organs and causes a number of diseases.

The Heart: The well-known cardiac symptoms in beriberi demonstrate that the cardiac muscle can be greatly injured by vitamin B_1 deficiency. Cowgill states: "Pure vitamin B_1 has no decided influence on the normal heart; only in the B_1-deficient organism does administration of the vitamin result in demonstrable effect." The effect of vitamin deficiency on the heart is manifested by tachycardia, aggravated by the least exertion, dyspnea, edema, right and left ventricular enlargement, and often apical or basal systolic murmurs. In cases of myocardial weakness associated with dilatation and signs of congestive heart failure, the addition of adequate doses of vitamin B_1 to the other cardiac therapeutic agents helps to correct the cardiac output and imparts a sense of well-being to the individual by removing the great fatigue they usually experience.

The Nervous System: The central nervous system, the autonomic system and the peripheral nerves show decided evidence of impaired function in vitamin B_1 deficiency. This is particularly true of the peripheral nerves as is evidenced in polyneuritis and beriberi.

The Digestive System: Anorexia, digestive disorders, such as flatulency, constipation, diarrhea or both, coated tongue or glossitis and various signs of malnutrition are fairly common in hypovitaminosis B_1. While the gastrointestinal manifestations are not specific for this deficiency they are nevertheless prominent findings

Diseases Caused by Vitamin B_1 Deficiency: The most important of the diseases caused by vitamin B_1 deficiency is beriberi. Other diseases such as neuritis and polyneuritis found during pregnancy, and cases of diabetes, pellagra, sprue, pernicious anemia, colitis and alcoholism may be associated with vitamin B_1 deficiency or may be ameliorated by the proper use of this vitamin

Beriberi is described as a deficiency disease due to the lack of vitamin B_1 in the diet. It is characterized by multiple neuritis, edema and cardiac weakness. Those who subsist exclusively or nearly exclusively on a diet of polished rice are subject to attacks of beriberi because vitamin B_1 is contained in the external layers of the rice which is completely removed by overmilling or polishing. Beriberi occurs in three forms: (1) Dry beriberi, in which the symp

disturbances. Nicotinic acid is a proven remedy for the successful treatment of pellagra, but has no effect upon the polyneuritis which may occur in pellagra. This phase is improved with the use of thiamin chloride. Nicotinic acid cures black tongue in dogs

Pellagra is classified as a deficiency disease due to avitaminosis of one of the B complex group and possibly to a lack of other substances vital to proper metabolism. Pellagra is found among those who are on a deficient diet, or have gastrointestinal disorders that interfere with the absorption of the material necessary for its prevention. In this country pellagra is found among some of the southerners who subsist largely on corn pone and molasses, and among the population of the entire country who are inveterate drinkers and keep themselves drunk for months at a time During their debauches their diet is restricted and during those intervals the gastrointestinal tract is so disturbed that it is incapable of absorbing the vitamins unless they are administered in concentrated form Persons on a strict, poorly chosen or fadist diet and the insane may also develop pellagra because of dietary insufficiency

Symptoms The disease is slow in onset, the prodromal period may be two or three months. During this stage there are vague digestive disturbances, loss of appetite, slight diarrhea, mental depression, headache, vertigo and insomnia Later there develop the characteristic skin lesions on the back of the hands, neck and face, chiefly over areas exposed to the sun. The lesions are generally symmetrical in location on the body and are sharply defined They start as an erythema and then darken; the skin may become hardened; vesicles, bullae or fissures may develop, and secondary infection may set in. The digestive symptoms are anorexia, stomatitis, glossitis, diarrhea and achylia gastrica. The nervous symptoms vary from functional neurosis to severe dementia and cord changes (SEE: Fig 3, p 134).

Treatment: Patients who were given riboflavin alone did not show complete recovery, while when nicotinic acid was added, or when nicotinic acid alone was administered, many of the pellagra patients were apparently cured.

Nicotinic acid alone or in conjunction with vitamin B complex appears to be an ideal method of preventing and curing pellagra and other deficiency diseases of that type An adequate amount of brewer's yeast with a diet rich in green vegetables, fruits, milk and liver will, because of the vitamin content, improve or cure this disease (SEE · pp 916, 918)

Dosage For prophylaxis, when on an insufficient diet or in a nonabsorptive state, 20 to 60 mg. can be given daily. For treatment where the disease has already developed, 100 to 1000 mg may be given in divided doses daily, it may be administered orally or parenterally

Other Vitamin B Factors: Vitamins B_3, B_4, B_5, and the W factor are still in the preclinical stage of study From the studies upon laboratory experimental animals, only this much may be said; that B_3 is a growth factor for pigeons; that B_4 is an antiparalytic factor as applied to chicks and rats; that B_5 is a weight maintenance factor for pigeons, and that factor W is a growth essential for rats Future studies of these factors may prove their values as nutritional substances in man

Vitamin B_6 (Pyridoxine): ***The Rat Antidermatitis Factor:*** Vitamin B_6 is found in fairly large quantities in maize (Indian corn), and it has also been pre-

in greater concentration than in any other part of the plant. In broccoli the leaves contain twice as much riboflavin as the flower, buds, or the twigs. It is excreted in fairly large amounts in the urine. Riboflavin has been synthesized and has a distinct chemical formula

Physiology: Goldberger and Lilly,[1] in studying pellagra, found that some of the animals on a deficiency diet developed a dermatitis in any one of the following parts: The ears; the front of the neck; the upper part of the chest; forearms; back of forepaws; shins, or the back of the hindpaws. Sebrell and Butler induced riboflavin deficiency in humans This was manifested by the development of mascerated areas at the angles of the mouth (cheilitis) which developed into transverse fissures The mucosa of the lips became shiny, almost red, and had a denuded appearance There were also greasy seborrheic accumulations at the alae nasae, around the eyes, and, in some instances, on the ears. These lesions disappeared after the patients were treated with riboflavin; nicotinic acid had no effect. It is therefore assumed that riboflavin is just one of the constituents of vitamin B_2, the absence of which may be partly responsible for some of the manifestations of pellagra but not for the entire syndrome; and that riboflavin is concerned with the development of lesions around the mouth and in the gastrointestinal tract in humans and may produce nutritional dermatitis in chicks and cataracts in rats. It is now believed that nicotinic acid and not riboflavin is the pellagra preventative vitamin.

Daily Requirement of Riboflavin: According to Rose, the daily requirement of Borquin-Sherman units of riboflavin to prevent deficiency manifestations are: In children up to ten years of age about 400 units or 20 units per 100 Calories, if more than 2000 Calories per day are consumed; in adults, also 20 units per 100 Calories. Steiperling recommends 450 units for boys under six and girls under seven years of age; 540 units for boys from seven to ten and girls from eight to 13 years of age; 600 units for older children and adults, or approximately 570 units per capita population In riboflavin deficiency, one to three milligrams of crystalline riboflavin given daily would correct the deficiency.

Unit: The unit of riboflavin (Borquin-Sherman unit of vitamin G) represents three to five micrograms of the substance. Others give it a higher value, eight to ten micrograms per unit, or the amount required for a rat to gain 40 Gm. weight in 30 days. One mg. (1000 micrograms) of riboflavin is equivalent to 400 (Borquin-Sherman) units of vitamin B_2 (G)

Source of Vitamin B_2: Liver-stomach concentrate is one of the most satisfactory sources of vitamin B_2. Riboflavin is synthesized from several substances (For availability and therapeutic use, see pp. 915, 916).

Nicotinic Acid or Nicotinic Acid Amide *(The P.P. Factor):* Nicotinic acid (amide) is identical with the P.P. factor and is one of the constituents of vitamin B_2. It is known chemically as pyridin-3-carboxylic acid. The deficiencies produced by an inadequate amount in the system are pellagra, alimentary disorders, such as proctitis (diarrhea), dermatitis, pigmentation and thickening of the skin, glossitis, stomatitis, urethritis, vaginitis and nervous and mental

[1] Goldberger, J., and Lilly, R. D.: Pub. Health Rep., 41: 1025, May 28, 1926.

known under the following names: Ascorbic acid, hexuronic acid or cevitamic acid.

The normal vitamin C content of the blood ranges from 0 8 to 1 8 mg. in 100 cc. Values of 0.3 mg. to the 100 cc. of blood are found in scurvy. The mean vitamin C blood concentration is about 1 mg. to the 100 cc The normal output of ascorbic acid in the urine is about 13 mg. daily.

Sources: Among the foods rich in vitamin C are oranges, limes, lemons, (raw and canned), tangerines, tomatoes, (raw or canned), fresh strawberries, black currants, green peppers, raw cabbage, properly prepared leafy green vegetables such as spinach, brussel sprouts, kale, broccoli, parsley and dandelion leaves Other important sources are onions, kohlrabi, cauliflower, turnips and beets Lettuce, endive and escarole have a lower vitamin C content Fruits, other than citrous type, such as apples, bananas, pineapples contain a lesser quantity of vitamin C, and dry cereals and the legumes are devoid of vitamin C However, almost any seed soaked in water for 24 hours and kept moist for a few days until it sprouts develops an effective antiscorbutic substance and retains it even when cooked.[1]

Among the animal products, liver is a fairly good source of vitamin C. Cooked meat muscle contains very little. Butter, eggs and cheese contain no vitamin C and pasteurized milk very little.

Physiology: Vitamin C has an effect upon the intercellular colloids and on the cells as a whole It influences favorably the red corpuscles, platelets and other blood elements; also the bone and the denture.

[1] Bessey, Otto A, Ph D: The Vitamins, A M A, 1939.

Defective intake or scarcity of vitamin C in the system will affect the *ends of the long bones* causing rarefaction of the cortex and various osseous changes The costachondral junctions become enlarged. The periosteum shows weakening of its attachment; the periosteal lesions are prone to be complicated by hemorrhages. The *teeth* become weakened and defects develop in the enamel and the dentine. The *gums* become swollen, ulcerated, and may become gangrenous, often causing hemorrhage. The *eyes* show ecchymosis and occasionally there may be other signs of eye trouble. The *skin* develops the characteristic scurvy lesions, follicular or petechial hemorrhages These hemorrhages are commonly noted in the lower extremities. The *viscera* also suffer from this deficiency, showing hemorrhages, and, occasionally, necrosis and ulceration. The adrenals usually atrophy.

The Unit: One mg. of ascorbic acid equals 20 U S.P. units. Orange juice, freshly prepared from fresh fruit, contains 13 8 U.S.P. units per cc.

Daily Requirements: It is estimated that infants require from 8 to 50 mg. daily; children from 22 to 100 mg. or more daily; adults from 30 to 100 mg. or more daily. During pregnancy and lactation, the quantity of vitamin C required is larger than at any other time

The individual requirement of vitamin C can be fairly accurately determined by one of three tests.

1. The resistance or fragility of the blood capillaries.

2. The excretion of ascorbic acid in the urine.

3 The ascorbic acid content in the fasting blood.

Pathology: A deficiency of vitamin C causes a number of diseased conditions.

pared synthetically in crystalline form. The administration of vitamin B_6 has failed to cure pellagra or black tongue, and likewise failed to prevent the occurrence of pellagra when given in conjunction with a pellagra-forming diet. However, Spies *et al*[1] report that they used pyridoxine for the treatment of several cases of pellagra and beriberi who suffered from nervousness, insomnia, irritability, abdominal pain, weakness and difficulty in walking He administered 50 mg. of pyridoxine in normal salt solution intravenously, and within 24 hours the patients were free of symptoms. Pyridoxine is said to cure the acrodynia-like dermatitis of rats. It is considered as a distinct entity belonging to the vitamin B complex group, and appears to be of nutritional value in man, but its exact rôle has as not yet been definitely proven.

Recently, reports have appeared in the literature citing the beneficial results obtained from the use of vitamin B_6 in pseudo-muscular dystrophy,[2] the nonencephalitis type of parkinsonism,[3,4] cheilosis,[5] and in arsenical polyneuritis when given in conjunction with vitamin E[6] (For availability and therapeutic use, see pp. 916, 918)

Pantothenic Acid (Filtrate Factor): This was first discovered by Williams and his co-workers[7] in 1933 as a powerful growth-stimulating acid found in various plants and in the tissues of animals, particularly in the liver. Rohrman, Burget and Williams[8] later found it to be a constituent of all types of cells and thought it to be vital for cellular respiration. In 1939, Williams[9] stated: "Pantothenic acid is a vitamin of importance in animal nutrition." Pantothenic acid was isolated in pure form by McHenry and Gavin[10] and it was synthesized by Woodley[11] in 1940. At present it is administered in conjunction with other vitamins in defective nutritional states. It has been estimated by Jukes[12] that the pantothenic acid requirement for the chick is something like 1.4 mg. per 100 grams of diet. Intensive studies are now being conducted to determine the rôle played by pantothenic acid in human nutrition (For availability and therapeutic use, see pp. 916, 918)

Avitaminosis B may be caused by a deficiency of one or more of the B complex group, due either to insufficient intake or deficient absorption and utilization of one, of several, or of the entire B complex group.

Vitamin C (Cevitamic Acid)

Vitamin C is the antiscorbutic vitamin (cevitamic acid). In human beings it is found in fairly large quantities in the adrenals and in the circulating blood. Szent-Gyorgyi isolated hexuronic acid (cevitamic acid) from the adrenals claiming that ½ to 1 mg daily of this substance will protect against scurvy

In food, vitamin C is found in abundance in citrous fruits and green vegetables. It is also synthesized and is

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[1] Spies, T D, Bean, W. B, Ashe, W. F · J A M A 112: 2414, 1939

[2] Antopol, W, and Schotland, C E.· J. A M A 114 1058, 1940

[3] Jolliffe, N H Minnesota Med 23: 542, 1940

[4] Spies, T D, Hightower, D P, and Hubbart, L H. J A M A 115 · 292, 1940

[5] Smith, S G., and Martin, D W, · Proc Soc Exper Biol and Med 43 660, 1940.

[6] Vilter, R W, Aring, C D, and Spies, T D J A M A 115 209, 1940

[7] Williams, R J, *et al* J Am Chem Soc 55 2912, 1933.

[8] Rohrman, E, Burget, G E, and Williams R. J · Proc. Soc Exper. Biol and Med 32: 473. 1934.

[9] Williams, R J: Science 89 · 486, 139

[10] McHenry, E W, and Gavin, G Science 91: 171, 1940

[11] Woodley, D W.· Science 91. 245, 1940

[12] Jukes, T. H: Biol. Chem. 129: 225, 1939.

tect which prevents the ultilization of calcium and phosphorus.

Vitamin E

Vitamin E is now recognized as the reproductive vitamin; it is derived from wheat germ oil as a *tocopherol* and has been synthesized It prevents or delays autoöxidation of fats and the resulting rancidity. It is also found in other vegetable oils, such as lettuce and in tomato, and is produced synthetically as tocopherol and ephynal.

It was found that when pregnant rats were kept on a diet poor, or a diet deficient in vitamin E, the embryos died and were resorbed

Vogt-Moller[1] reported that he injected 20 cc. of sterilized wheat germ oil in otherwise normal cows who had failed to become pregnant Following the injection, pregnancy occurred in 33 out of 50 instances Other experiments have shown that the administration of large doses of wheat germ oil has increased the size of rabbit litters, reduced the mortality of suckling pigs, and when wheat germ oil was added to the hen's food, it increased the hatchability of eggs. It was reported by Wagenen that cellular changes take place in the anterior lobe of the pituitary body of vitamin E deficient male animals Hypoplasia of the thyroid was found in vitamin E deficient adult rats and cretinism in vitamin E deficient young rats

In the human female it was found that a deficiency in vitamin E will diminish the blood supply and the nutrition of the embryo; and in the male it will cause liquefaction of the chromatin material in the spermatozoa and spermatids and prevent spermatogenesis. Currie[1] reported that by administering 3 minims of wheat germ oil daily from the beginning of pregnancy in women who had the "abortive habit," he secured 23 normal births out of 24 cases. Threatened abortion and premature separation of the placenta were prevented by the use of wheat germ oil.

Wechsler[2] reported encouraging results obtained in early cases of amyotrophic lateral sclerosis treated with synthetic vitamin E ("Ephynal," Roche).

Cases are reported where carcinoma developed after the prolonged administration of an impure wheat germ oil. Vitamin E appears to have a beneficial effect upon the reproductive organs. However, more intensive study is necessary before it can be intelligently included among the useful vitamins

Vitamin E Unit: The vitamin E unit is as yet not definitely established. Each gram of wheat germ oil (Lilly) contains approximately two Evans-Burr units of vitamin E (For availability and therapeutic use, see pp. 917, 919).

Vitamin K

Vitamin K is known as the antihemorrhagic or coagulation vitamin. The numerous reports in the literature concerning vitamin K testify to its efficacy in preventing and in stopping certain types of hemorrhage caused by a prothrombin deficiency.

Sources: Vitamin K is probably formed in the body and not taken in with the usual food as are the other vitamins It is believed that vitamin K is synthesized by the action of putrefaction bacteria in the intestinal canal from which

[1] Vogt-Möller, P · *Acta path et microbiol., Scandinav*, 12. 115, 1935

[1] Currie, D. W: Brit. Med J, 1·752, April, 1936

[2] Wechsler, I S · J A. M A, 114: 948, 1940

normal balanced diet. When on a "reducing diet," vitamin D should be added.

For Women: Women require 0 55 Gm. or more since extra calcium is lost during menstruation. During pregnancy and lactation women require extra amounts of calcium; this may be supplied by giving vitamin D and about three times the usual quantity of calcium. This may be obtained from 7000 units of vitamin D or 1.5 Gm. of calcium.

Infants: Breast-fed babies require less vitamin D than do artificially-fed infants.

Growing Children: Those children who are not on a rich calcium diet, or who are unable to metabolize calcium and phosphorus because of diarrhea or other defects, should receive from 300 to 400 units of vitamin D In rickets, the amount of vitamin D required may be from 100,000 to 500,000 units or more daily.

It is to be borne in mind that vitamin D is not a substitute for calcium, it only facilitates the proper utilization of calcium and phosphorus that are in the body.

Physiology and Pathology of Vitamin D: Vitamin D is considered the *antirachitic vitamin*; it both prevents and ameliorates rickets, and cures it if treatment is begun before permanent changes have occurred. It has a definite effect upon rachitic bone structure, calcium and phosphorus metabolism, and also upon phosphatase and other metabolic processes. Vitamin D facilitates the absorption of calcium and phosphorus and probably diminishes its excretion from the bowel. It bears some relation to the parathyroids since it influences calcium and phosphorus metabolism. However, their actions differ in many respects. Vitamin D produces healing of the metaphyseal lesions of rickets, while the parathyroid hormone may retard it. Both, however, will relieve tetany.

Hypervitaminosis D: The administration of excessive doses of vitamin D will cause hypercalcemia, increased density of the epiphyseal ends of the bones with rarefaction of the shafts. The calcium phosphorus balance becomes negative. Calcific deposits occur in the tubules of the kidneys, blood vessels, heart, stomach and other organs. Diarrhea, vomiting and other gastrointestinal defects, as well as certain nervous manifestations may become evident.

Hypovitaminosis D: In severe cases there will develop rickets, extreme nervousness, twitchings, convulsions and tetany. Milder manifestations of vitamin D deficiency are hypocalcemia of various degrees associated with hyperphosphotemia

The need of calcium may be determined by examination of the ends of bone, by x-ray study of the bones and by chemical determination of the calcium-phosphorus content of the blood.

The Use of Vitamin D in Diseases Other Than Rickets: Vitamin D has been used in the treatment of tetany, nervous irritability, atrophic arthritis, psoriasis, urticaria, mucous and ulcerative colitis, tuberculosis, osteomalacia, and a host of other conditions, but its efficacy has not as yet been proven.

It is well to bear in mind that a properly balanced diet during health will supply the necessary requirement of vitamin D and that sunshine is nature's method of supplementing any deficiency that may exist in the diet.

A deficiency of vitamin D may be due to improper diet, insufficient sunshine or to some intrinsic metabolic de-

amount of bile or bile salts must be given simultaneously.

Indication for Vitamin K Therapy: Vitamin K is indicated in the hemorrhagic diseases of the newborn, in the bleeding of the various types of jaundice, providing the prothrombin level is below normal. It is, therefore, useful in hemolytic icterus, certain types of hepatocellular disease, in biliary fistula, when bleeding occurs following biliary tract operations and also as a preoperative prophylaxis in cases of liver and gallbladder disease.

Clark, Dixon, Butt and Snell[1] list the following conditions in which vitamin K is useful:

"The fat soluble vitamin K is useful in the treatment of prothrombin deficiencies which occur in other conditions besides jaundice

"The proper absorption and utilization of the antihemorrhagic food factor depends on the following conditions: (1) The diet must contain the antihemorrhagic factor, (2) bile of normal composition must be present in the intestinal tract, (3) proper digestion of fat is necessary, (4) a sufficient amount of normal intestinal mucosa for the absorption of the substance is required, and (5) a normal liver is essential

"Hemorrhage sometimes occurs in cases of postoperative intestinal obstruction in which transduodenal aspiration is carried out for a long time, thus removing most of the bile from the intestines Such hemorrhages can be prevented by the administration of vitamin K and bile salts.

[1] Clark, R L, Dixon, C F, Butt, H R, and Snell, A M Proc Staff Meeting, Mayo Clinic (June 28, 1939) Review of Gastroenterology, 6 451 (Oct) 1939.

"In cases of both external and internal fistula there may be lack of an adequate mucosal surface for absorption of vitamin K and a prothrombin deficiency produced.

"Chronic ulcerative colitis may cause prothrombin deficiency due to rapid transit of food through a canal in which the absorptive mucosal area has been decreased by disease.

"A decrease in prothrombin may also occur in patients with faulty digestion of fat, as in nontropical sprue

"The authors recommend vitamin K therapy in cases of intestinal obstruction, intestinal fistula, gastric retention and in continuous duodenal aspiration"

Vitamin K is of no benefit in hemophilia, purpura (thrombocytopenia), aplastic anemia, acute leukemia, the hemorrhage from telangiectasis, gastric or duodenal ulcer, pulmonary tuberculosis, and ruptured blood vessels, because in these conditions the prothrombin levels are normal

Vitamin K Unit: A definite unit has as yet not been determined The dose of vitamin K is variable There does not seem to be any fear of inducing a hypervitaminosis K (SEE: pp 917, 920)

Dosage: Snell[1] suggests (1) Patients having normal prothrombin levels and requiring only prophylactic measures, should be given alfalfa concentrates with bile capsule orally

(2) Patients with definitely prolonged clotting time may be started on oral therapy If the response is inadequate, they should be given liquid extracts with bile salts by way of the duodenal tube

(3) Patients actually bleeding should receive blood transfusions in addition to

[1] Snell, A M, *et al* Proc Staff Meet, Mayo Clinic, 13 753 (Nov 30) 1938

it is absorbed and stored somewhere in the body, *possibly in the liver.* Several substances that possess antihemorrhagic properties have been isolated from various sources and chemically identified. These are known as K_1, K_2, phthiocol, and several others.

The early work of Dam and Schonheyder and of Almquist and Stokstadt has shown that chicks fed on a certain *diet developed hemorrhagic disease which* was not cured by any of the then known vitamins, *i. e*, A, the B's, C, D, and E, *but the addition of alfalfa cured or pre*vented the hemorrhagic disease

Vitamin K is at present considered as a fat soluble *substance found in fairly* large quantities in alfalfa, in decomposed fish meal, and also in hemp seed, the fats of hog's liver, chicken liver, and human feces. It is obtained for clinical use in a watery and oily solution from alfalfa and fish meal.

Physiology and Pathology of Vitamin K: Vitamin K stops or prevents hemorrhage by raising the prothrombin in the blood. Hemorrhages not due to a low prothrombin level are not influenced by the administration of vitamin K. A. J. Quick[1] has shown that the sweet clover disease of young cattle, and the bleeding of other animals fed on a vitamin K poor diet, were caused by a low prothrombin level in the blood This is cured by feeding alfalfa. By this observation and the observations of others it seems fairly certain that vitamin K is essential for the synthesis of prothrombin in man, dog, rat, chickens and other animals.

The Rôle Played by Prothrombin in Blood Coagulation: According to the theory of Schmidt, Feld and Morawitz, prothrombin in the presence of calcium is transformed by the enzyme thromboplastin (thrombokinase) liberated from injured tissue or thrombocytes (platelets) to thrombin. Thrombin reacts with fibrinogen to form fibrin, thus causing clotting. A low prothrombin level in the blood interferes with blood coagulation, causing prolonged clotting *time.* Excessive *doses of vitamin K* do not decrease the clotting time in the normal. Heparin, a substance which *delays clotting, acts on the thrombo*plastin (thrombokinase), while vitamin K speeds clotting by increasing the pro*thrombin. The two substances* are not antagonists, since each acts upon a different factor of the coagulation mechanism.

Since the only type of hemorrhage controlled by vitamin K is a low prothrombin level, it is necessary to determine the prothrombin level in the blood before vitamin K is given, unless the case be one of jaundice, or of injury to the liver or bile ducts.

The Owen and Hoffman method for determining the approximate prothrombin blood level is as follows: 10 cc. of venous blood is placed in a test tube with an excess of thrombokinase, and the exact clotting *time is noted.* This is compared with the blood-prothrombin solution of a known normal *subject The ratio between the* two is known as the clotting activity; variations below 100 per cent indicate a bleeding tendency. When the clotting activity is less than 50 per cent, hemorrhages may occur.

Vitamin K increases clotting (stops hemorrhage) only in the presence of bile salts. When vitamin K is given either by mouth or parenterally, an adequate

[1] Quick, A. J.: Am. J. Physiol., 118:260 (Feb) 1937.

vitamin A and D concentrate of cod-liver oil is available for intramuscular use in 1 cc. ampoules, each containing 13,200 units of vitamin A and 1884 units of vitamin D.

Carotene (the previtamin A substance) is available in tablets, and in capsules (as carotene in oil), also as carotene with vitamin D concentrate in oil, and as cod-liver oil with carotene and vitamin D concentrate.

Vitamin B: This is a complex vitamin containing several factors, each having its distinctive chemical formula and therapeutic action, though they complement one another. Vitamin B complex occurs in abundance in brewer's yeast, which is the most potent method of administration of the entire B group. Brewer's yeast is obtainable in solution. One or two teaspoonfuls is to be given once or twice daily or oftener when necessary. Brewer's yeast is also available as a dry powder, and as tablets, plain or coated, and in capsules. The vitamin content of each of the constituents is marked on the package.

Vitamin B_1: This is the antineuritic vitamin. It has been synthesized as thiamin and is dispensed as thiamin chloride or more properly, as thiamin hydrochloride.

Chemical Formula: $C_{12}H_{17}N_4OClS\ HCl$.

Food Sources: Yeast, whole grain cereals and breads, liver, chicken, pork and nuts, etc.

Therapeutic Use: Thiamin chloride is employed in the treatment of beriberi, the neuritides (especially of alcohol), pellagra and anorexia. It is also used as an addition to diets poor in vitamin B_1 content and in those on high carbohydrate diets, and as an aid in stimulating the appetite and optimum growth in infants and children. It is claimed to have beneficial effects in myocarditis, in exophthalmic goiter, during pregnancy in general debility, in multiple sclerosis, in polyneuritis and in herpes zoster. It is of definite benefit in irradiation sickness when given in doses of about 10 mg. intravenously daily, or every other day, until improvement is noted. Thiamin chloride should be administered intravenously in doses from 1000 to 10,000 U.S.P. units for the acutely ill or in severe cases where rapid response is desired. In the more chronic or in the milder cases when the digestive tract is capable of absorption, vitamin B_1 may be administered orally, alone or in combination with other required vitamins.

The Unit: One milligram is the equivalent of 333 U.S.P. units.

Daily Requirement: The average daily requirement of U.S.P. units for adults is 200 to 300; for infants, 50 to 75.

Availability: Thiamin chloride is available in powder, tablet, and liquid form for oral use and in ampoules and vials in an aqueous solution for intramuscular and intravenous use. It is also available in various combinations with other vitamins and with various substances as tablets, pearls, pills, capsules, syrups and elixirs. Each of the preparations lists the vitamin content.

Vitamin B_2 or G: This is known as riboflavin, lactoflavin, ovoflavin or flavin, and is considered as the anticheilosis vitamin. It is prepared synthetically.

Chemical Formula: $C_{17}H_{20}N_4O_6$

Food Sources: Yeast, liver and milk.

Therapeutic Use: Riboflavin is indicated in cheilosis, glossitis, lesions on the sclera and cornea, in general malnutrition and, in conjunction with vitamin B_1, in beriberi, pellagra and multiple sclerosis. The dose is 1 to 5 mg. daily

the duodenal *instillation of vitamin* K and bile.

Preparations containing vitamin K activity in oil have been given intramuscularly in association with the oral administration of *bile* or *bile* salts Watery and dry preparations are given by mouth. The daily requirement is uncertain. Several *synthetic preparations* having vitamin K hemostatic properties are now available (SEE: p. 920).

Biotin was formerly classified as vitamin H. It is now grouped as a member *of the B complex. Deficiency of biotin* in the diet of chicks will cause scaly dermatitis. It is believed that biotin plays an important rôle in the synthesis of lipids.

Inositol *is another addition to the B* complex group. Experimentally it was capable of curing alopecia in mice and "spectacled eye" in rats.

Résumé of Better Known Vitamins, Their Therapeutic Uses and Available Products

Avitaminosis is a state in which there is usually a deficiency of several vitamins, *and in which one of the groups* may be predominatingly deficient Since the vitamins are complementary to one another, it is often advisable to employ several vitamins simultaneously. In pronounced *cases of a specific vitamin* deficiency, the deficient vitamin alone may be used.

Mode of Administration: In some cases supplying the vitamin-containing food in abundance may correct the defect. If, however, the individual is incapable of ingesting a *sufficient quantity of the* required food, or if the condition of his digestive apparatus is such that it cannot absorb or utilize the vitamins from his ingested food, then it becomes necessary *to administer the required* vitamins in sufficient amounts either to prevent avitaminosis or, if present, to cure it

The vitamins *may* be administered orally when the absorptive capacity of the digestive apparatus is capable of absorbing them, otherwise, many of them may be administered intramuscularly and several *of them intravenously*. For quicker response, the intravenous route, when possible, is preferable; otherwise the intramuscular route may be chosen

Vitamin A: Antixerophthalmic vitamin.

Chemical Formula. It has been isolated in crystalline form as $C_{20}H_{29}OH$.

Food Sources Butter, leafy vegetables, carrots, liver and egg yolk, etc

Therapeutic Uses. Vitamin A is employed in the treatment of night blindness, xerophthalmia and hyperkeratosis when due to vitamin A deficiency, also in debilitated states of children and adults. *It helps to promote growth and* increases bodily resistance to certain infections. It is suggested that it may prevent the formation of certain types of kidney and gallbladder stones; it has *also been used in hyperthyroidism.* In the presence of jaundice, vitamin A is not absorbed from the intestinal canal.

The Unit· The U S P or International Unit is 0 6 microgram of carotene or 0 3 *microgram of vitamin* A.

Daily Requirement (U S P. or International Units): *Adults, 3000 to 8000;* children, 6000 to 10,000, during preg-

as *cod-liver oil and halibut* liver oil, in tablets, pearls and in oily bulk and with viosterol. It is also dispensed *in* combination with other vitamins. The unit content is marked on the package. A

	Properties and Sources	Method of Standardization	Definition of Unit	Deficiency Manifestations		Estimated Daily Requirement
				Clinical-Laboratory Tests	Clinical Signs and Symptoms	
C Ascorbic Acid	Water-soluble, white crystals Stable to acids, destroyed by alkalies Citrus fruits, beet greens, broccoli, etc Produced synthetically	Chemical titration using 2 6 dichlorophenol—indophenol or iodine Biological prophylactic tests with guinea pigs	U S P (also International) unit is equal to 50 micrograms of 1-ascorbic acid	Assay of blood and urine for vitamin C which is decreased Saturation test Roentgenologic study of long bones which show evidence of subperiosteal hemorrhages	Subclinical deficiency characterized by weakness, increased irritability, anorexia, pyorrhea, poor wound healing and perifollicular hemorrhages. Scurvy shows petechial hemorrhages, swelling and bleeding of the gums, loosening and loss of teeth, pain and swelling of the extremities	Children, 600-1000 U S P. units. Adults, 1000-3000 U S P units.
D	Fat-soluble Pro-vitamin for D_2 is ergosterol and for D_3, 7-dehydrocholesterol Certain fish liver oils eggs, beef and pig liver contain natural vitamin D_3	The U S P method is concerned with the cure of rickets in rats	U S P (also International) unit is the antirachitic activity of one milligram of a standard solution of purified irradiated ergosterol in oil equal to 0 025 microgram of crystalline vitamin D (calciferol)	Roentgenologic examination for abnormal calcification of bones Determination of serum phosphatase Determination of phosphorus and calcium	Delayed bone growth, defective tooth structure and rickets in infants and children Tetany in infants osteoporosis in adults	Children, 400 to 1000 U S P. units Adults not known, but 300-400 U S P units is probably an adequate daily intake
E Alpha-Tocopherol	Fat-soluble, exists naturally as a yellow oil. Wheat germ oil, leafy vegetables and cereals Produced synthetically	The amount of material necessary to bring about normal gestation in a vitamin E deficient rat and also chemical and colorimetric methods of testing for tocopherols	There is no recognized unit Potency is expressed as the amount (or as equivalents) of alpha-tocopherol present per gram or cubic centimeter	None	Vitamin E therapy apparently offers some promise in the treatment of muscular dystrophy and in prevention of threatened or habitual abortion in women Its use in functional sterility has been disappointing	Daily requirement uncertain but vitamin E appears to be essential at least for certain physiologic processes connected with muscle metabolism and reproduction
K_1	Vitamins K_1 and K_2 occur in nature and are fat-soluble Certain synthetic vitamin K compounds are water-soluble Produced synthetically Alfalfa, spinach, bacteria	Determination of the amount of vitamin necessary to restore normal coagulation time in the blood of vitamin K depleted chicks	No U S P or International unit A chick curative unit is equal to 0 5 microgram of 2-methyl-1, 4-naphthoquinone	Determination of plasma prothrombin clotting time which is abnormally prolonged	Tendency to abnormal bleeding due to hypoprothrombinemia	Daily requirement is uncertain.

"*Therapeutic Notes*"—Parke, Davis & Company, Sept 1941, Detroit, Mich.

	Properties and Sources	Method of Standardization	Definition of Unit	Deficiency Manifestations		Estimated Daily Requirement
				Clinical-Laboratory Tests	Clinical Signs and Symptoms	
A	Essentially colorless, fat-soluble, destroyed by oxidation Certain fish liver oils, butter, green leafy plants	Measurement of growth response in vitamin A *deficient rats by U S P* method Physical measurement with a spectrophotometer, based upon light absorption at 3280 Angstrom units	U S P (also International) unit is the growth-promoting activity of 0 6 microgram of pure beta-carotene	Biophotometer test for subnormal dark adaptation Analysis for blood carotene and vitamin A	Night blindness xerophthalmia keratinization of skin and mucous membranes with increased susceptibility to infections entering by way of epithelial structures retardation of growth in children	Children, 6000-8000 U S P units Adults, 3000-6000 U S P units. Pregnant and lactating women, 6000-8000 U S P units.
B_1 Thiamin *Hydrochloride*	Water-soluble white crystals, unstable at high temperatures and in alkaline media Stable in acid media Yeast, rice polishings, cereal grains Produced synthetically	The U S P method is concerned with the cure of polyneuritis in rats Colorimetric chemical tests and microbiological methods have also been devised	U S P (also International) unit is equal to three micrograms of crystalline vitamin B_1	*Determination* of thiamin excretion in urine Assay of blood for bisulfite binding substances which are usually increased	*Anorexia, neurasthenia, calf muscle* tenderness constipation peripheral neuritis, edema, tachycardia on slight exertion, flattening of T wave in electrocardiogram	*Children,* 100-600 *U S P* units. Adults, 300-600 U S P units.
B_2 Riboflavin	*Water-soluble, yellow* crystals, heat-stable, destroyed by light Milk, eggs, liver Produced synthetically	*The determination of the* growth response of vitamin B_2 deficient rats to riboflavin Microbiological method employing *Lactobacillus casei* also used	No U S P or International unit Expressed as the equivalent weight (in micro*grams) of riboflavin One* Sherman-Borquin unit is equal to 2 5 micrograms	Determination of urinary excretion of riboflavin	Cheilosis, seborrhea involving the nasolabial folds ears and face, magenta colored glossitis vascularizing keratitis, impaired growth, lack of vigor	Daily requirement is uncertain but may be 1 to 4 milligrams for growing children and adults, requirements of *pregnant* and lactating women are probably approximately 50 per cent higher
Nicotinic Acid	Water-soluble, white crystals Liver, lean meats, milk, eggs, peanuts, beans, peas, tomatoes Produced synthetically	Biological tests on dogs with black tongue Assays with microorganisms and chemical methods	No U S P or International unit Expressed as the weight (in milligrams) of nicotinic acid or nicotinamide per gram or cubic centimeter	*Determination* of nicotinic acid and coenzymes I and II in blood and urine which are usually decreased in clinical pellagra	Fiery red tongue, stomatitis, diarrhea and abdominal distention, ptyalism, mental disturbance, erythema, desquamation, and pigmentation of ex*posed parts of body* and about the genitals	Daily requirement is uncertain, but probably ranges between 15 and 25 milligrams for adults and older children, 5 to 10 milligrams daily for infants and young children (under 10)
Pantothenic Acid	Water-soluble, white crystals, stable to heat and to oxidizing or reducing agents Liver, brewer's dried yeast and eggs Produced synthetically as the salt	Biological growth methods using *either rats or chicks* and a microbiological method which measures the growth of *Lactobacillus casei*	No U S P or International *unit* Expressed as the weight of pantothenic acid (in micrograms) per gram or cubic centimeter	Assay of blood and urine for pantothenic acid	None known at present but it *seems* likely that further clinical studies will indicate that certain manifestations of B complex deficiency as encountered in pellagra and ariboflavinosis will be shown to be caused by pantothenic acid deficiency	*Daily* requirement unknown but pantothenic acid is essential to normal health.
B_6 Pyridoxine Hydrochloride	Water-soluble, white crystals stable to heat, acids and alkalies. Yeast liver Produced synthetically	The determination of the growth response of de*pleted rats* to pyridoxine Colorimetric chemical test	No U S P or International unit Expressed as the weight of pyridoxine (in micrograms) per gram or cubic centimeter	Colorimetric tests of urine Excretion studies following intravenous injection of a test dose of pyridoxine hydrochloride	Certain symptoms of pellagra that are *unresponsive* to thiamin nicotinic acid and riboflavin have been reported to improve following treatment with vitamin B_6 Used experimentally in management of Parkinsonism and the neuromuscular dystrophies	Daily requirement unknown but pyridoxine is apparently required for normal health

dental caries, pyorrhea and certain gum infections; also in anorexia, anemia, malnutrition due to vitamin C deficiency and in various infections and post operatively. It has also been used with apparent success in rheumatic fever, arthritis, lead poisoning, osteomyelitis, whooping cough, hemorrhagic diseases, delayed wound healing, drug sensitivity and ulcers It should not be dispensed in alkaline solutions or in combination with alkalies The average oral dose for mild or moderate cases is 50 to 150 mg daily. In severe cases it may be given 0 5 to 1 Gm intravenously.

The Unit· 1 mg. represents 20 U S P. units

Daily Requirement The average daily requirement of U S P units is· 500 to 2000 units, depending upon weight, *i e.,* 8 to 32 units per kilogram (2.2 lbs) of body weight.

Availability· When possible, it may be adequately administered as fresh orange juice. One ounce (30 cc) of orange juice contains about 17 mg of cevitamic acid. Ascorbic acid or cevitamic acid is obtainable in powder form and in 25, 50 and 100 mg tablets for oral use It is also obtainable in vials for intravenous use

Vitamin D: Antirachitic factor.

Chemical Formulae· Vitamin D_2 (calciferol) $C_{28}H_{43}OH$

Vitamin D_3 (7-dehydrocholesterol) $C_{27}H_{43}OH$

Food Sources The usual foods, except those mentioned, do not contain appreciable amounts of vitamin D It is found in abundance in the livers of cod, halibut, shark, and, to a lesser extent, in other fishes, *i e.,* salmon, sardines and herring Milk, eggs, and meat products contain calcium and also traces of vitamin D Vitamin D milk is a fortified milk.

Therapeutic Use: Vitamin D is employed for the prevention and treatment of rickets, of spasmophelia and of osteomalacia, and for influencing a favorable calcium and phosphorus balance wherever necessary. It is often used as a routine during infancy, childhood, pregnancy and lactation. It has been used with apparent favorable results in tuberculosis, scrofula, inanition, celiac disease, arthritis, psoriasis, dental caries and locally in various skin lesions and ulcers.

The Unit· The U.S P. Unit: This is essentially the same as the International Unit. It is the activity of one milligram of an international standard solution of irradiated ergosterol (viosterol). The minimum standard for cod-liver oil is at least 85 U S P. units of vitamin D per gram. Viosterol should contain up to 10,000 U.S.P. units of vitamin D per gram

Daily Requirements· Not definitely determined; varies with age, sex, etc

Availability· Cod-liver oil, halibut liver oil and in combination with vitamin A in viosterol, procurable in bulk, in capsules, pearls and tablets; also in irradiated milk.

Note· The precursors of vitamin D are ergosterol, 7-dehydrocholesterol and other sterols.

Vitamin E: *α- Tocopherol; also β- and* γ-Tocopherols (Antisterility Vitamin)

Chemical Formula· Formula of synthetic alphatocopherol, $C_{29}H_{50}O_2$.

Food Sources· Whole grain, lettuce, wheat germ oil, cottonseed oil, palm oil, rice oil, etc

Therapeutic Use: While the use of vitamin E is still experimental, it is being used in threatened abortion, sterility, defective spermatogenesis, muscular dystrophy, amyotrophic lateral sclerosis

The Unit: The U.S.P. unit is 1 microgram (0.001 mg.). The Sherman-Bourquin unit is approximately 2.5 micrograms. One mg. (1000 micrograms) is the equivalent of 400 Sherman-Bourquin units.

Daily Requirements. The average daily requirement is from 400 to 750 Sherman-Bourquin units, depending upon age.

Availability: Riboflavin is obtainable in one or more milligram capsules and in combination with other vitamins in capsules or tablet form for oral use.

Nicotinic Acid: This is known as the pellagra-preventative (P.P.) vitamin. It is prepared synthetically as pyridine3-carboxylic acid (amide).

Chemical Formulae: Nicotinic acid $C_6H_5O_2N$; nicotinic acid amide $C_6H_6ON_2$

Food Sources: Liver, wheat germ, yeast, etc.

Therapeutic Use: Nicotinic acid, nicotinic acid amide and sodium nicotinate are all effective, or are specific, in the treatment of pellagra. Nicotinic acid has also been used with apparent success in alcoholic psychosis of the Korsakoff type, in the initial syndrome of pellagra characterized by hyperesthesia and increased psychomotor and emotional drives, in xerostomia, in Ménière's disease and in sulfanilamide cyanosis. The dose for pellagra is 500 or more milligrams in divided doses of 50 mg. daily. The intravenous dose is 10 to 15 mg. four to five times daily. Larger doses may cause peripheral vasodilation. Nicotinic acid amide is less likely to cause the unpleasant sensations experienced from the use of nicotinic acid.

The Unit: Expressed in milligrams.

Daily Requirement: Approximately 20 to 60 mg.

Availability. Nicotinic acid is obtainable in powder and in tablets for oral use, 25, 50 or 100 mg. per tablet, and in solution for intravenous use.

Vitamin B_6 Pyridoxine (Acrodynia Factor): This has been synthetically prepared as pyridoxine hydrochloride.

Chemical Formula: $C_8H_{11}O_3N.HCl$

Food Sources: Maize, whole cereals, liver, cane molasses and yeast.

Therapeutic Uses. Pyridoxine in conjunction with other vitamins, appears to be of value in subnutritional states. It has been used with apparent success in Parkinsonism (not the postencephalitic type); in the pseudomuscular dystrophies; in arsenical polyneuritis (in conjunction with vitamin E); in cheilosis, and in the macrocytic type of anemia.

The Unit: Expressed in micrograms.

Daily Requirements: Not definitely determined.

Availability. Pyridoxine hydrochloride is available in 1 and 25 mg. tablets for oral use and in 2 cc. ampoules containing 50 mg. in isotonic solution.

Pantothenic Acid (Filtrate Factor of B Complex; Antidermatitis Factor): Pantothenic acid and calcium pantothenate in doses of 3 mg. three times daily (orally) is being used with some measure of success for premature graying, especially of young individuals.

Vitamin C: Known as cevitamic acid or hexuronic acid. It is the antiscorbutic vitamin. It has been prepared synthetically.

Chemical Formula: $C_6H_8O_6$

Food Sources: Oranges, lemons, limes, grapefruit, tomatoes, cabbage, water cress, fresh strawberries and other leafy vegetables and berries.

Therapeutic Use: Cevitamic acid is employed in the treatment of scurvy,

CHAPTER XXX

Allergy, Its Clinical Manifestations and Diagnosis

The subject of allergy has awakened new interest in medicine, particularly since the clinical manifestations of the various allergens have become better known and the reactions of sensitive individuals have been more carefully studied Allergic reactions are specific in that certain substances will affect certain individuals in a definite way.

An *allergic reaction* may be defined as the sensitized host's method of protesting against the invasion of an unwelcome guest. The entrance of the offending guest may have been affected through the skin, the mucous membrane, the respiratory system, the gastrointestinal system or directly through the blood The allergic manifestations are many and varied, depending upon the host's sensitivity. These may be enumerated as headache, migraine, rhinitis, conjunctivitis, bronchitis, asthma, nausea, vomiting, cramps, diarrhea, cardiac palpitation, urticaria, eczema and other skin rashes, arthralgia, etc

The *allergens* (substances causing allergic reactions) are likewise many and varied All types of food, plants, trees, grasses, pollens, animal emanations, dander, feathers, wool, dust, bacteria, fungi, and practically everything with which we ordinarily come in contact may give some persons an allergic reaction which may be manifested in some part of the body

Anaphylaxis and Allergy: Anaphylaxis in animals closely resembles allergy in man Indeed some of the protein sensitization phenomena produced in man by the injection of sera or other substances closely resembles the anaphylaxis in animals. There is, however, sufficient difference between anaphylaxis and allergy to warrant a description of each.

Anaphylaxis is a term applied to induced hypersensitization in animals. It is defined as an exaggerated reaction of an animal to the second dose of the protein by which it was previously sensitized. For example, if 0 1 cc. of horse serum is injected into an animal, and 10 to 14 days later a larger amount, say 1 0 cc., is again injected in the same animal, severe shock or death will occur within a comparatively short time as the result of the second injection.

Allergy, or *allergic reactions*, is a term applied to somewhat similar reactions in human beings. Dorland defines allergy as "a condition of unusual or exaggerated specific susceptibility to a substance which is harmless in similar amounts for the majority of members of the same species." Allergic sensitivity appears to be an hereditary tendency manifesting itself spontaneously on exposure to specific substances While chemically and physiologically there seem to be a number of differentiating points between anaphylactic reactions in animals and severe allergic reactions in men, clinically the difference is not very obvious. We have seen severe shock and an occasional death induced in humans after the injection of antitoxic horse, cattle or rabbit serum. At present, before serum is injected into a patient, his sensitivity to that type of serum is tested by intradermal injection or conjunctival instillation of a minute dose of that

and certain other cord lesions. Dose: 2 to 4 cc. or more daily.

The Unit U.S.P. unit not standardized.

Daily Requirements. Not definitely determined

Availability: As wheat germ oil in bulk and pearls. Trade names: Zygon (Squibb); Ephynal Acetate (Roche), Tocopherex (Squibb), etc

Vitamin K: Coagulation or prothrombinogenic factor This has been synthesized.

Chemical Formulae: Vitamin K_1 (2-methyl-3-phytyl-1, 4 naphthoquinone) $C_{31}H_{46}O_2$.

Vitamin K_2: $C_{41}H_{56}O_2$.

Vitamin K-Analogs (2 methyl-1, 4 naphthoquinone): $C_{11}H_8O_2$.

Food Sources: Alfalfa leaf and meal; hog liver; hempseed; cabbage, spinach, tomatoes, etc

Therapeutic Use Vitamin K and K-active substances are employed to prevent and stop hemorrhage due to prothrombin deficiency. Employed in hemorrhagic diseases of the newborn, in the bleeding of jaundice, and preoperatively to prevent hemorrhage, and postoperatively to stop hemorrhage in patients with jaundice and liver derangements; also in intestinal conditions where the absorption of vitamin K from the intestines is defective and in various liver diseases associated with impaired utilization of vitamin K Vitamin K and K-active substances are valueless in the treatment of purpura hemorrhagica, hemophilia and other hemorrhage not due to prothrombin deficiency.

The Unit: Not yet standardized.

Daily Requirements: Not definitely determined.

Availability: Vitamin K is obtainable as natural vitamin K or as one of the synthetic products which are very effective. Both are dispensed in capsules, tablets or in vegetable oil solution for oral and intramuscular or subcutaneous use. An aqueous solution is prepared for intravenous use The dose depends upon the conditions and may vary from 1 to 15 mg. or more daily. The oral use is preferred wherever possible. Vitamin K, whether natural or synthetic, to be effective must always be administered in conjunction with bile or bile salts.

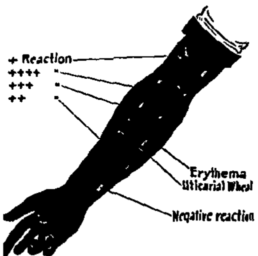
Trade Names. Vitamin K Concentrate, Klotogen, Proklot, Naphthoquinone, Thyloquinone, Quino-Thrombin, Hydro-quinone, etc

Vitamin P (Citrin, Eriodictyol) is found in citrous fruits in close association with vitamin C. It is believed to be a factor in capillary fragility. A lack of vitamin P in the system will cause fatigue, pain in the legs and shoulders accompanied by petechial hemorrhages. The hemorrhages caused by vitamin P deficiency differ from those caused by vitamin C deficiency. In the former, there are small petechiae in the skin, while in the latter, the hemorrhages are large and occur in the subcutaneous tissue and muscle.

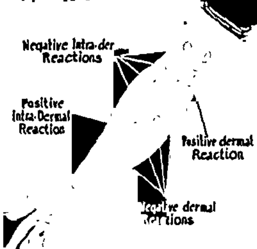
Calcium eriodictate, 100 to 150 mg, was given orally daily by Rappaport and Klein[1] to 12 children with capillary fragility They were cured in six months

Para Aminobenzoic Acid: This is now considered to be a vitamin belonging to the B complex group. It is believed to be a growth factor in chicks, and also appears to be an achromotrichia factor Rats who became gray on a deficient diet when given p. aminobenzoic acid returned to black. Its use for humans is still in the experimental state.

1 Rappaport, G H and Klein, S. J Pediat 18 321, 1941



Dermal Tests



Illustrating Dermal and Intradermal Tests

(Balyeat's "Allergic Diseases," F. A. Davis Co., Philadelphia, Pa.)

specific serum If the patient shows specific sensitivity, he is desensitized by slowly injecting a small portion of the serum subcutaneously over a period of hours to which may be added fractional doses of adrenalin chloride. If the patient is acutely sensitive to serum, such treatment should, if possible, be withheld.

Desensitizations: This term denotes a method of treatment by which the individual's tolerance is raised to a substance to which he is allergic, hypersensitive or intolerant.

For example, if a person is sensitive or allergic to a specific food, minute quantities of that food are given at infrequent intervals; as the tolerance increases, ascending quantities of the food are given at more frequent intervals until tolerance is established. Similar procedures are carried out with other allergens. In hay fever, the patient receives subcutaneously, ascending doses of the pollen to which he is allergic in advance of the "season" so that when pollinization takes place, the patient's tolerance has been sufficiently raised so that his allergic reactions are either less severe or, rarely, are nonexistent.

Etiology: Allergic reactions may become manifested during infancy, childhood, or during adulthood. In many instances, sensitivity to certain foods, pollens and other substances is traceable as a familial peculiarity; in others no family taint is discernible. Just why an adult who has lived in the same surroundings, has eaten the same type of food all his life, and has in no way changed his habits, occupation and mode of living should suddenly become hypersensitive to objects with which he came in intimate contact throughout his entire existence is not easily explainable on the theory of previous sensitivity The fault may lie in some change in the chemistry of the individual, and not in the substances with which he comes in contact. If it were due to contact substances, then all persons coming in contact with these substances would develop similar symptoms. It is not beyond the realm of possibility that allergy may be the expression of a deficiency disease, or that it occurs because of disease or disturbance of some "center" in the body whose function it is to stabilize the vasomotor mechanism of the body. Amelioration of symptoms by the process of desensitization does not strike at the underlying cause; it only relieves or smooths over symptomatic manifestations. Desensitization is not unlike the application of an icebag in a febrile disease.

Clinical Manifestations of Allergy

The commonest symptoms of allergy are found in the eyes, nose, respiratory system, digestive system, skin, nervous system, cardiovascular system, and in the blood.

Allergic Manifestations of the Eyes

Conjunctivitis: This is the commonest of the eye disturbances seen in allergy. It occurs in hay fever, and often is the only sign of pollen sensitivity. The symptoms are redness, injected vessels, tearing, itching and, at times, photophobia Other substances such as food, drugs, exposure to sera, bacterial products, dust, to strong sunlight and to heat may, in sensitive individuals, be the cause of conjunctivitis. It is often important to differentiate allergic conjunctivitis from catarrhal conjunctivitis caused by foreign bodies, by infections, by irritating vapors and

by eyestrain. Occasionally it becomes difficult to determine whether a certain substance, such as mascara, causes conjunctivitis because of allergic sensitivity or because of its irritating quality. A good rule to remember is that in allergic conjunctivitis there are other signs of allergy such as rhinitis, headache and eosinophilia accompanying the reaction and that minutely ascending doses of the irritant applied to the eyes has a tendency to decrease the severity of the conjunctivitis. Also, when the "irritant" is applied to one eye and both become inflamed, it is most likely an allergic reaction. Infections and irritants usually produce an inflammation in the exposed eye alone

Vernal conjunctivitis, because of its seasonal appearance, is believed by some to be an allergic manifestation; others believe that it is just a local reaction of the eyes to heat.

Cataract: Particularly in the young, this has been ascribed in some instances to allergic sensitivity. Such patients will show hypersensitivity to lens proteins or to other proteins

Exposure to Light: This will, during the spring and summer, cause allergic phenomena which are characterized chiefly by sneezing. We have noted quite a number of people who, when they get out into the sunlight in the morning, get a paroxysm of sneezing, usually four to ten times; when this is over they do not sneeze any more until the next morning. The sneezing attacks may be prevented when the eyes are shaded with dark glasses for the first hour during exposure to the sun

Allergic Manifestations of the Nose

Allergic Rhinitis: This may be caused by the same provocative agents that cause allergic conjunctivitis. The symptoms may be local and confined to the nose, or general in which the rhinitis is only one of a number of manifestations, as in hay fever.

Symptomology: Allergic rhinitis is a distinct entity. Its symptoms are similar to other forms of rhinitis, though the etiology may be varied. During the attack the patient breathes through the mouth; speech is nasal or has that peculiar quality found in those who suffer nasal obstruction. In addition to this, there will be noted in many cases a profuse, thin watery discharge trickling uncontrollably through the nares. In some cases the discharge indicates added infection. Sneezing may come on spontaneously, after physical effort, after meals, on change of posture or when the mucous membrane of the nose is irritated. Inspection of the nasal chambers will reveal a bluish-gray, glistening, somewhat pale mucous membrane covered with a thin or mucoid secretion The turbinates are swollen, appear engorged or edematous and polypoid growths frequently add to the discomfort of the patient Examination of the nasal secretion, by staining a "smear" in the same manner as a differential blood smear, will show a large number of eosinophils.

Etiology: Allergic rhinitis may occur seasonally resulting from the inhalation of pollens during spring (tree or rose fever) or during autumn (hay fever). It may also occur perennially or at infrequent intervals caused by certain foods, drugs, bacterial agents, animal emanations and all the other agents that may cause local or general allergic phenomena in sensitized individuals.

Differential Diagnosis: Allergic rhinitis or coryza is to be differentiated

changes, wearing apparel, overexposure to sun rays, x-rays and also physical allergy such as exhaustion and nervous excitability may initiate an attack of asthma. An attack may be of short duration or it may last for weeks at a time with periods of remissions and exacerbations. The attacks may come on during the day or night, depending upon the causative factors.

Symptomatology: The general symptomatology of asthma depends upon the length of time the individual has had it. During the early stages, the condition can only be diagnosed during an attack or from the patient's descriptions. The symptoms are severe paroxysmal dyspnea of the expiratory type, accompanied by wheezing (sibilant râles) and frequent short dry coughs. Chronic cases or those who have had frequent attacks of asthma for years will show definite constitutional changes These are emphysematous chest, signs of chronic emphysema, enlarged heart, distended vessels, and signs of chronic bronchitis. During attacks, the dyspnea may be more severe and is accompanied by general cyanosis, distention of the superficial veins, severe cough with some expectoration; and intermingled with the attacks of dyspnea or orthopnea there are periods of suffocation or strangulation due to the patient's inability to get air into the lungs. In very chronic cases there may be associated sinusitis, peribronchial fibrosis, bronchietasis and clubbing of the finger tips.

The expectoration may be profuse and thick, or it may be scant, it may contain various microorganisms as secondary invaders. Other microscopic findings in sputum are Charcot-Leyden crystals, Curschmann's spirals and eosinophils.

Differential Diagnosis: Allergic asthma is to be differentiated from other types of asthma. Asthma due to lung encroachment such as pneumoconiosis, tumor, abscess, tuberculosis, bronchiectasis and chronic bronchitis may be diagnosed by the physical findings in the lungs, the constancy of the dyspnea and the excessive cough with expectoration. *In these conditions physical exertion will cause first cough and expectoration and then dyspnea; physical* exertion will increase the dyspnea, and the cough is in the nature of an explosive expiration so as to free the lungs from as much air as possible. Cardiac asthma is really not asthma but orthopnea due to left ventricular failure. These attacks come as a rule during the night; the dyspnea is more of the inspiratory type; the râles are both of the dry and of the moist varieties; there is considerable cyanosis, and definite signs of myocardial failure

Hay Fever: The name "hay fever" is a misnomer, since generally in this disease there is no fever and it is not caused by hay. Usage of the term hay fever has, however, identified it with a definite syndrome. Therefore hay fever may be defined as a seasonal allergic reaction characterized by:

(1) Acute *conjunctivitis such as* burning, redness with itching and tearing of the eyes.

(2) Acute coryza manifested by itching and running of a thin discharge from the nose, with frequent and paroxysmal sneezing spells.

(3) Dry irritating cough.

(4) In severe cases, asthmatic attacks.

"Hay fever" is a seasonal allergic symptom complex depending upon the specific sensitivity of the individual to

from acute rhinitis or coryza due to infection. In bacterial infection the onset is slower than in allergic rhinitis; the nasal secretion is thicker and often excoriates the nares, the nasolabial fold or the upper lip. There may be an associated rise in temperature, headache and other manifestations of an "acute cold" The coryza preceding an infectious disease such as measles, typhus fever, etc, is easily diagnosed with the appearance of symptoms of that disease. In acute coryza the mucous membrane of the nose is red and inflamed and the turbinates may be swollen; in the subacute or chronic condition there may be associated sinus infection Examination of the nasal discharge will show a high neutrophil count in inflammatory rhinitis, and a high eosinophil count in allergic rhinitis. It is to be borne in mind, however, that an individual who has or has had chronic rhinitis may develop an allergic rhinitis, or one with allergic rhinitis may develop an inflammatory rhinitis, making the differential diagnosis difficult.

Allergic Manifestations of the Respiratory System

The allergic phenomena referable to the respiratory system were known long before other allergic manifestations were recognized. The most prominent of these phenomena is bronchial asthma

Bronchial Asthma: This may be defined as a syndrome characterized by attacks of expiratory dyspnea During the attack there are short inspiratory efforts followed by prolonged pauses which are followed by prolonged and difficult expirations. As the attack continues, the inspirations also become labored because of the attempt to force air into the lungs which are overfilled with air that should have been expelled by the preceding expiratory effort. During these attacks the accessory muscles of respiration are brought into play. Many sibilant râles of varying pitch are heard during respiration, most numerous during expiration. These are caused by the air being forced through the partially constricted lumina of the smaller bronchi and bronchioles.

Asthma may result from a number of causes such as allergic manifestations, cardiac disease, bronchiectasis, tuberculosis and other inflammatory or space taking lesions in the lungs or bronchi. The mechanical cause of asthma is a constriction of the air passages which prevents an adequate interchange of air in the lungs. In allergic asthma the offending pathology is a spastic contraction of the smaller bronchi and bronchioles Whether this contraction is caused by the direct action of the allergen on the bronchial musculature and mucosa or directly upon the vagus which causes the bronchial phenomena is not definitely known. Asthma may occur at all ages The first attack may be initiated during infancy, childhood, adolescence, adulthood and even in old age.

Etiology: There is no question that asthma is a familial disease; occasionally however it is not traceable to any kin, though other members of the family or clan may show allergic manifestations other than asthma, such as urticaria, eczema, hay fever, etc.

The exciting factors of allergic asthma are those that may excite allergic manifestations elsewhere, though the sensitivity of the respiratory tract is greater than of any other part of the body. Pollen, dusts, vapors, foods, drugs, bacteria and their products, animal emanations, dander, feathers, temperature

testinal cramps, diarrhea, constipation and occasionally hemorrhage.

Caution: Before a definite diagnosis of allergy of the gastrointestinal tract is made a thorough gastrointestinal study should be done by a physical examination of the abdomen and the rectum; a chemical and microscopic examination of the stomach and bowel contents and x-ray examination of the entire gastrointestinal tract including the gallbladder is important A person may show a definite allergic sensitivity to food, and at the same time may have an organic lesion or a parasitic infection somewhere in the digestive system which may be overlooked by taking "allergy" for granted It should also be borne in mind that most of the systemic diseases and infections cause gastrointestinal disturbances

Allergic Manifestations in the Skin

The allergic manifestations of the skin are many and varied; these may appear in conjunction with other signs of sensitivity, or they may appear alone The various skin manifestations may be caused by the ingestion of certain foods or by contact with certain substances

The allergic skin phenomena (allergic dermatoses) are urticaria, angioneurotic edema, erythema multiforme, erythema nodosum, atopic dermatitis (eczema) and contact dermatitis of allergic type

Urticaria (Hives): Urticaria occurs as superficial swellings that are red and have a pale central area. These lesions are evanescent and may spread to various parts of the body and cause intense itching. The lesions may be small and confluent, causing welts, or they may be large and isolated

Etiology. Heredity plays a part The exciting causes are albuminous foods such as eggs, milk, shellfish, meats and occasionally other foods, fruits and berries. Autointoxication, gastroenteritis, constipation and other conditions in which there is an excessive production of histamin may usher in an attack Other substances that may cause urticaria are sera, antitoxins, drugs, inhalants (pollens), bacterial and parasitic infestations, external irritants and at times it may be due to nervous or psychic influences

Giant urticaria is a variant of urticaria. It involves both the superficial and deeper structures of the skin; there is usually marked itching and burning The lesions are larger and appear isolated, though large areas of the skin surface may be occupied by them

Angioneurotic Edema: This is a type of urticaria that involves the subcutaneous tissue and causes tumorlike masses upon the skin and mucous membrane of the face or other parts of the body. When the larynx or pharynx become involved it may cause suffocation

Erythema Multiforme: This consists of polymorphous exudative, bright red or dark red macular papulae or urticarial bulbous or hemorrhagic lesions distributed upon the face, the neck, the forearms, legs and dorsal surface of the hands and feet, and occasionally upon the mucous surfaces

Etiology: It is believed that the condition is caused by sensitization of the small cutaneous blood vessels by a variety of toxic or allergic substances to which some individuals are sensitive

Prurigo: This is a chronic itching papular affection which occupies chiefly the lower abdomen, buttocks and the extensor surfaces of the limbs It is believed to be an allergic manifestation

Eczema: Eczema during *childhood* has been proven in many instances to

certain types of pollens When not exposed to the specific pollen, even though it be the "hay fever season," no hay fever symptoms will occur. On the other hand, when exposed to the specific pollen, though out of season, allergic phenomena will become manifested.

The pollens responsible for hay fever are not the same for every hay fever sufferer. Some are sensitive to timothy or June grass, etc. (spring type), others to rag weed, saga brush, etc The flora differ in various countries and in various sections of one country. The United States has been roughly divided into six regions, each being characterized by the abundance of certain types of pollenizing plants which grow sparcely or not at all in the other regions.

Allergic Manifestations in the Digestive System

Since a great variety of foods and drugs have been proven to cause general allergic manifestations such as rhinitis, asthma, urticaria, etc, it is expected that these articles should also cause local gastrointestinal manifestations in sensitive individuals Yet the number of proven cases of purely local gastrointestinal allergic manifestations compared to manifestations elsewhere is rather small. It appears that many of the allergic manifestations caused by food depend upon the integrity of the digestive system It is not always the kind of food that the person ingests that is responsible for the reactions; it is the products produced during digestion that may cause allergic symptoms. Thus it is found that certain articles of food may cause allergic reactions at one time and not at another. Also, when an individual is skin tested for various foods it is often found that certain foods may give a severe skin reaction while there may be no reaction when they are ingested, even in large quantities. *Per contra*, other foods may give negative skin reactions but will at times cause severe constitutional reactions when ingested.

Symptoms: The gastrointestinal allergic manifestations may be divided into local and general symptoms

In the *mouth* these may consist of large or small, single or multiple ulcerations of the mucous membrane of the lips, cheeks, tongue or pharynx, which may be accompanied by mild paresthesia or partial anesthesia of these parts. The lesions are usually temporary.

In the *esophagus* there may develop local swellings which may cause difficulty in swallowing and substernal oppression. It is quite possible that the Vinson-Plummer's syndrome may be an allergic manifestation.

In the *stomach* the manifestations may consist of pylorospasm and occasionally of hypochlorhydria Tuft[1] cities several cases of gastric ulcer whose etiology is attributed to allergic manifestations.

Colon: Attacks of various types of nonspecific colitis such as mucous colitis, spastic colitis and possibly also ulcerative colitis have occasionally been recognized as being the result of allergic manifestations.

Rectum: Pruritus ani, multiple anal fissures, and tenesmus are not infrequently traceable to an allergic reaction to some food or to underwear that comes in intimate contact with the anus. Among the *gastrointestinal* symptoms caused by allergy are pain and burning of the mouth and tongue, nausea, vomiting, in-

[1] Tuft, Louis "Clinical Allergy," W. B Saunders Co., p 413, 1937.

testinal cramps, diarrhea, constipation and occasionally hemorrhage.

Caution: Before a definite diagnosis of allergy of the gastrointestinal tract is made a thorough gastrointestinal study should be done by a physical examination of the abdomen and the rectum; a chemical and microscopic examination of the stomach and bowel contents and x-ray examination of the entire gastrointestinal tract including the gallbladder is important. A person may show a definite allergic sensitivity to food, and at the same time may have an organic lesion or a parasitic infection somewhere in the digestive system which may be overlooked by taking "allergy" for granted. It should also be borne in mind that most of the systemic diseases and infections cause gastrointestinal disturbances.

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Etiology: Heredity plays a part. The exciting causes are albuminous foods such as eggs, milk, shellfish, meats and occasionally other foods, fruits and berries. Autointoxication, gastroenteritis, constipation and other conditions in which there is an excessive production of histamin may usher in an attack. Other substances that may cause urticaria are sera, antitoxins, drugs, inhalants (pollens), bacterial and parasitic infestations, external irritants and at times it may be due to nervous or psychic influences.

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Eczema: Eczema during childhood has been proven in many instances to

be due to some allergy. The lesions first appear upon the face as an erythema in which subsequently develop small epidermal vesicles; these rupture and produce moist and crusted areas. In the *adult*, allergic eczema may occupy the antecubital and popliteal fossae, the front and sides of the neck, the forehead and the areas about the eyes. Occasionally it may occupy other parts of the body.

Etiology: Food, clothing or other substances are often found to be the allergic factor in sensitive individuals.

Purpura: Cases of Henoch's purpura have been traced to individuals who exhibited other allergic phenomena. *Peliosis rheumatica* also belongs to the allergic group. In both conditions the etiology is attributed to allergic reactions of the skin to the bacteria causing these conditions.

Contact Dermatitis: This is the name applied to a group of skin eruptions caused by direct contact with the offending substances such as metals, dyes, drugs, foods, plants, various materials handled in occupations, etc.

Allergic Manifestations of the Nervous System

Migraine: This heads the list among the neurologic symptoms caused by allergy. While in quite a number of patients no definite proof of allergic sensitivity can be discovered, there are nevertheless a large number of patients suffering from migraine who show distinct sensitivity to a variety of allergens, chiefly foods It is at times difficult to trace the offending food substance since the reaction may be delayed for several days. It is believed that when food has undergone an atypical reduction in the gastrointestinal tract certain substances are there formed which cause the allergic reaction.

Simple Headache: This may also occur as an allergic manifestation.

Ménière's Disease and Idiopathic Epilepsy: Occasionally these respond to desensitization in persons who have shown strong allergic reactions when skin tested for certain substances. In such individuals it is believed that if allergens are not the primary cause of the disease, allergy is a strong contributing factor.

Allergic Manifestations in the Cardiovascular System

Thromboangiitis obliterans, coronary disease, angina pectoris, paroxysmal tachycardia, sinus tachycardia, extrasystoles and periarteritis nodosa have been found to be associated with other allergic phenomena, or have often been found in persons who are generally classified as allergic individuals. Whether or not allergy plays a prominent part in the causation of these affections awaits further study.

General Diagnosis of Allergy

While an individual may show clinical allergic manifestations, the specific allergen responsible for the phenomena cannot be diagnosed clinically. In order to identify the specific substance responsible for a reaction, various "skin tests" are required. A positive reaction is identified by a large erythematous areola in the center of which is a fairly large bleb showing pseudopodia

The differential diagnosis between such manifestations as may be due to allergic reactions and those caused by organic disease should be made by eliciting a careful and complete history, by making a thorough and systematic physi-

cal examination and by performing such laboratory tests as the conditions indicate might be helpful in the diagnosis. In other words, irrespective of the complaints, every patient should have a thorough examination.

Test for Protein Sensitization

The diagnosis and treatment of certain dermatological and respiratory conditions has been improved by the application of the theory of protein sensitization, and as the tests to determine these conditions are very simple, and their application so useful in general practice it seems advisable to include something concerning them.

Technic: The examiner makes a slight scarification upon the flexor surface of the patient's left arm or other convenient location, and rubs into it a small quantity of the suspected protein, or 0.1 cc of a properly prepared protein is injected intradermally so that it causes an elevation of the skin It is advisable to make a second scarification or intradermal injection some little distance from the first (the other arm is a suitable location), into which no protein is injected, this serving as a control and gauge of the degree to which the skin reacts to scarification alone

If the patient is sensitive to the protein employed, in from 15 to 20 minutes a marked wheal will appear at the place of scarification or of injection, the size of the wheal and the length of time it persists being indicative of the degree to which the patient is sensitized to the test protein Frequently the same patient will prove to be sensitive to several different proteins It is, of course, necessary to make a separate scarification or injection for each separate protein, which often cannot be done at one sitting, especially in nervous patients, or young children.

Tests in Hay Fever and Asthma: The chief use of the protein sensitization tests up to the year 1920 was in establishing the proper therapy of hay fever and asthma. A great deal of this work was done by William Scheppegrell of New Orleans, who has given especial study to the geographical distribution of the pollen-bearing plants which are the principal causal agents of these respiratory affections. An important feature is the fact that hay fever is due to atmospheric pollens, and that only these are needed for testing and immunizing purposes. Goldenrod is often mistakenly blamed for hay fever. It is, however, to be borne in mind that the most brilliant bloom of the goldenrod, *solidago canadensis*, is in October, when practically all of the hay fever attacks have subsided by the end of September (Scheppegrell)

"In making the diagnostic tests for hay fever, we are guided in the selection of the pollen extract, by the location It is, therefore, necessary to know the hay fever plants to which the patient is exposed, the representative biological group being sufficient in most cases East of the one hundredth meridian, we must test for the grasses, ragweeds and chenopods West of this meridian, the tests should in addition include the artemisias. The ragweed test should also be made in the Rocky Mountains and Pacific States, since, although the ragweeds are uncommon, there are other members of the ragweed or Ambrosiacae group, such as gaertneries, marsh elders, Iva, and cockle burrs, which respond to the same test and similar immunizing methods."

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TABLE III[1]

EXPECTATION OF LIFE AT SPECIFIED AGES FOR *TOTAL PERSONS (WHITE AND COLORED)* AND FOR WHITE PERSONS BY SEX, UNITED STATES, 1938

Age	Expectation of Life, Years		
	Total Persons	White Males	White Females
0	62 78	62 12	66 20
1	64.86	64 31	67 84
2	64 30	63 72	67 23
3	63 52	62 93	66 43
4	62 66	62 08	65 56
5	61 77	61 19	64 66
10	57 13	56 57	59 98
15	52 44	51 89	55 22
20	47 91	47 33	50 57
25	43.50	42 86	46 02
30	39 14	38 40	41 51
35	34 81	34 01	37 04
40	30 61	29 75	32 64
45	26 54	25 66	28 34
50	22 64	21 78	24 17
55	18 98	18 16	20 19
60	15 49	14 79	16 42
65	12 34	11 78	13 01
70	9 64	9 20	10 04
75	7 31	6 99	7.47
80	5 43	5 26	5.43

[1] Statistical Bulletin, Metropolitan Life Insurance Co, 21 5, 1940

to the situation prevailing in 1901, almost half of the white male babies would *have died before attaining age 57*, while the halfway mark on the basis of health conditions at present is at about 67 years For white females the corre*sponding ages were 61 years in 1901 and* about 72 years at present

Comparison of mortality rates in 1919 1929 and 1939 show that in ages 1 to 4 the 1939 death rate showed a decrease of 61 7 per cent since 1929 and 84 2 per cent since 1911, in ages 25 to 34 the decrease was 44 1 per cent since 1929 and 67 2 per cent since 1911, in ages 55 to 64, 19 6 per cent since 1929 and 31 6 per cent since 1911. In those over 75 there has been very little change in the mortality rate, a decline of only 2 8 per cent since 1911 (Table III). These statistics suggest the question: Do the older people individually attain a greater age, *i.e.*, do more people survive to 80 or 90 years or older? It is evident that the increase in the life expectancy has increased the *total number of older individuals, but* we are as yet unable to tell whether the span of the individual's life will be further increased among those born since 1901, *i. e.*, since Preventive Medicine *became more generalized.*

Notwithstanding the authentic statistical studies that there are more old people living at present than there were a generation or two ago, one often hears the remark made by persons between the ages of 40 and 60 that they recall having seen many more old people during their childhood than they see now The rea*son for such statement is obvious* When one is 10 or 12 years old, every person above the age of 40 appears to be senile Moreover, two generations ago, a person at the age of 50 or even younger not only appeared older than does one of that age now, but he really was more senile

At present, when eyesight begins to fail, it is corrected by glasses; when the teeth decay or fall out, they are replaced by artificial denture; and even hearing is improved by special appliances. The progress of Medicine has made the detection and eradication of gastric ulcer, gallstones and other gastrointestinal diseases comparatively easy. The anemias, syphilis and other chronic diseases are better controlled now than they were two generations ago Among the other factors that tend to make people appear younger are the increase in leisure due to shorter hours of work and labor-saving devices in shop and home, etc.;

CHAPTER XXXI

Geriatrics—Senescence and Diseases of Old Age

Aging is a natural phenomenon The number of aged is proportionately increased when the death rate during infancy and youth is reduced. The great increase in the life expectancy from birth to death during the past 35 years does not bear a definite relation to actual longevity. If the mortality rate before 1911 had been as low as it is now, there would have been many more old people living then than there are today, because the birth rate was very much greater At present the definitely declining birth rate is partially compensated by the decline in the death rate among infants and young people so that there are comparatively more people of the older group.

TABLE I[1]

EXPECTATION OF LIFE AT BIRTH AMONG TOTAL PERSONS (WHITE AND COLORED) AND AMONG WHITE PERSONS BY SEX, UNITED STATES, FROM 1901 TO 1938

Year	Expectation of Life at Birth, Years		
	Total Persons	White Males	White Females
1938*	62 78	62 12	66 20
1937*	61 48	60 75	65 08
1936*	60 81	60 18	64 36
1935*	61 37	60 72	64 72
1934*	60 79	60 24	64 18
1933*	61 26	60 86	64.40
1932†	61 07	60 69	64.38
1931†	60 26	59 88	63.56
1929–1931§	59.50	59 31	62 83
1919–1920¶		55 33	57.52
1910‡	51 49	50 23	53 62
1901‡	49.24	48 23	51 08

[1] *Statistical Bulletin, Metropolitan Life Insurance Co*, 21 5, 1940.

* Total United States

† United States, excluding Texas.

‡ Original Death Registration States

§ United States, excluding Texas and South Dakota.

¶ Aggregate of 27 States, not computed for total persons

General and Individual Longevity

According to statistical studies published by the Metropolitan Life Insurance Company in 1940, (Table I, 1901-38) the average length of life or expectation of life at birth for white males is 62.12 years, and for white females 66 20 years, and for total persons (white and colored) 62.78 years In 1901 the life expectancy for white males was 48 23, and of white females 51.08 years, indicating an increase of almost 14 years for males and 15 years for females during the past 38 years Of this increase a greater amount has taken place in the ten years from 1928 to 1938 than in any similar previous period, namely, an increase of 6.65 years.

The life expectancy at various ages from birth to 80 is given in Table II

An even more striking evidence of the improvement in longevity since the beginning of the century than that provided by the expectation of life at birth is found in the proportions of the babies born who survive to later years of age In 1901 less than nine out of every ten white male babies born alive survived to reach their first birthday. At present health conditions have improved to such an extent that at least nine out of every ten newly born attain age 24. Among white girl babies, too, less than nine out of every ten born in 1901 survived their first year of life; now nine out of every ten babies will reach age 32. According

TABLE IV

CRUDE DEATH RATES PER 100,000 FOR PRINCIPAL CAUSES.[1] ALL AGES 1911, 1929, 1934, AND 1939

Causes of Death	1939*	1934	1929	1911†
All Causes of Death	760.9	854.1	934.2	1253.0
Typhoid fever	.7	1.5	2.4	22.8
Communicable diseases of childhood	4.2	11.1	20.2	58.9
Measles	.6	2.7	3.0	11.4
Scarlet fever	.7	2.6	2.7	13.1
Whooping cough	1.6	3.7	5.7	7.1
Diphtheria	1.3	2.1	8.8	27.3
Influenza and pneumonia	52.7	76.4	130.5	131.1
Influenza	9.8	11.4	41.9	15.9¶
Pneumonia—all forms	42.9	65.0	88.6	115.2¶
Tuberculosis—all forms	44.9	59.4	86.9	224.6
Tuberculosis of respiratory system	40.4	52.2	76.7	203.0
Syphilis, locomotor ataxia, and general paralysis of the insane	11.1	12.3	12.6	11.0
Cancer—all forms	103.1	96.1	77.6	68.0
Diabetes mellitus	27.5	24.4	18.3	13.3
Alcoholism	1.2	2.3	3.4	4.0
Cerebral hemorrhage, apoplexy‡	59.7	63.2	58.0	64.2
Diseases of heart§	160.5	162.9	146.8	141.8
Diseases of the coronary arteries	40.2	18.8	**	**
Angina pectoris	6.3	10.0	9.1	3.9
Diarrhea and enteritis	5.4	11.1	20.8	27.9
(under 2 years)	3.7	8.1	16.2	13.0
Appendicitis	10.2	13.1	14.0	10.9
Chronic nephritis (Bright's disease)	51.4	64.9	69.4	95.0
Puerperal state—total	5.4	8.8	13.6	19.8
Total external causes	59.3	73.2	80.3	97.9
Suicides	8.6	9.5	8.5	13.3
Homicides	4.4	5.9	6.6	7.2
Accidents—total	46.3	57.8	65.2	77.4
Accidental burns	2.4	3.6	5.0	8.8
Accidental drowning	4.2	5.3	6.4	10.2
Accidental traumatism by fall	9.6	11.1	9.0	13.2
Accidental traumatism by machines	.8	.8	1.6	1.8
Railroad accidents	2.2	2.6	3.9	9.5
Automobile accidents	17.1	21.1	21.0	2.3
All other accidents	9.9	13.3	18.3	31.6
Other diseases and conditions	119.1	144.6	170.3	257.9

1 Statistical Bulletin, Metropolitan Life Insurance Co, 21 1, 1940 (Figures taken from Industrial Department)

Incidence of Morbidity: As to the question of morbidity among the older group, we may definitely state that, while the mortality rate has decreased, the morbidity rate has increased. There are two main reasons for the present increase in the morbidity of the aged group. First, before the advent of Preventive Medicine, it was largely a matter of the "survival of the fittest." Only those who were endowed with unusual powers of resistance and were physically fit survived the ravages of infantile and youth diseases; therefore during old age they were constitutionally sound and did not as readily develop the degenerative diseases to which the less hardy are subject. Secondly, those who survived the various infectious diseases and epidemics during their youth developed an immunity which protected them against these infections and their sequellae in after years.

At present, since many weaker individuals, by means of Preventive Medicine and better general medical care, have been kept alive to reach old age, the incidence of morbidity in the senile group is naturally greater Both because of the increased number of old people and the fact that many of them are constitutionally inferior, the rate of mortality from degenerative diseases (cardiovascular, cerebrospinal and renal diseases, diabetes, etc) is greater than it was be-

* All 1939 death rates are subject to slight correction, since they are based on provisional estimates of lives exposed to risk.

† *Ages 1 and over only*

‡ Rates for 1930 to 1939 are not strictly comparable with those for earlier years, due to changes in classification procedure.

§ *Excluding pericarditis, acute endocarditis, acute myocarditis*, coronary artery diseases, and angina pectoris

** Included in all other diseases and conditions prior to 1930.

¶ Not comparable with the rates for 1929 to 1939.

TABLE III[1]

DEATH RATES PER 100,000 FOR ALL CAUSES OF DEATH. TOTAL PERSONS, BY AGE PERIODS 1911, 1929, AND 1939*

Ages	1939*	1929	1911	Percent Decline 1939 Since	
				1929	1911
One and Over	751 0	891.9	1,253 0	15 8	40 1
1–4	233.5	609.5	1,479 1	61.7	84 2
5–9	102.7	221.8	416 2	53 7	75.3
10–14	91.2	166.6	268 0	45.3	66 0
15–19	152.1	315.7	467 8	51.8	67.5
20–24	212 3	445.1	732 5	52.3	71 0
25–34	311.2	556 9	947.7	44.1	67 2
35–44	559.1	866.8	1,367.8	35.5	•59.1
45–54	1,152.1	1,555.7	1,978.3	25.9	41.8
55–64	2,461 4	3,061.5	3,596 0	19.6	31.6
65–74	5,575 6	6,505 0	7,455 0	14.3	25.2
75 and Over	13,536 7	14,283 4	13,926 9	5.2	2 8

* All 1939 death rates are subject to slight correction, since they are based on provisional estimates of lives exposed to risk

[1] Statistical Bulletin, Metropolitan Life Insurance Co, 21 : 1, 1940 (Figures taken from Industrial Department)

better hygiene and health habits; the increased popularity of outdoor recreation, athletics, vacations, etc. The dressmaker, clothier and beautician have also added to the youthful appearance of older people. Today a person at the age of 50 or even older sees well, hears fairly well, has better digestion, is more interested in his surroundings and looks better than did his grandparents' generation at the same age. Because of these, the person at 50 or 60 now not only looks but is a very much younger individual than was the person of equal age half century or more ago.

Onset of Old Age: There is always a question as to when old age begins Many medical authorities and poets alike consider old age as the Autumn of life and place its beginning at 60 years. There are obviously many exceptions. Moreover there are as many Spring and Summer days during Autumn as there are cold wintery days. So in the human, many may show advanced deterioration years before they have reached their fiftieth year, and others may fail to show such changes for years past their sixtieth birthday.

Process of Aging: Aging is not always a uniform process. There are some individuals who show the effect of age in their somatic structures while the mentality remains clear and comparatively young. Such individuals are among the unhappiest because they cannot understand why their bodies can no longer perform the duties which their minds dictate. Others show mental deterioration while their bodies are comparatively young These individuals are quite happy since they are not conscious of their limitations. The happiest seniles are those whose somatic and mental processes age simultaneously since their minds and bodies docilely accept their infirmities.

aged; they may occur in association with cardiac, renal, hepatic and prostatic disease.

Paralytic ileus, intestinal obstruction and strangulated hernia are serious accidents in the aged

Cholelithiasis often becomes manifested past the age of 50; at times gallstones may be silent Cirrhosis of the liver in the aged may be the result of infection or irritation by toxic substances suffered at an earlier age.

Symptoms of indigestion are common among the aged and may not necessarily be due to organic disease. Indigestion in the senile may be due to faulty diet, improper mastication, anemia, diminished gastric and intestinal secretions, diminished tonicity of the gastrointestinal tract, viceroptosis, passive congestion or circulatory failure

The Nervous System: Moore[1] classifies the neurologic conditions encountered after the age of 50 as follows:

I. ***Vascular disorders*** such as cerebral thrombosis, cerebral hemorrhage (localized, spreading and disruptive and intraventricular hemorrhage), cerebral embolism, hypertensive encephalopathies, and cerebral arteriosclerosis (focal and diffuse manifestations)

II ***Intracranial space taking lesions*** such as primary brain tumors (glioma, meningioma and other forms), metastatic malignancy, abscess, subdural hematoma, tuberculoma, gumma

III ***Degenerative diseases*** such as senile psychosis, Alzheimer's disease, Pick's disease, Schilder's disease, multiple sclerosis, combined sclerosis (pernicious anemia), Parkinson's disease (idiopathic and postencephalitic).

[1] Moore, M T The Penna Med Jour, 44 195, 1940

IV. ***Inflammatory disease*** such as syphilis (meningovascular, paresis, tabes dorsalis and other forms), meningitis (epidemic, acute purulent, tuberculous), encephalitis.

V. ***Miscellaneous conditions*** such as pellagra, migraine, intoxications (alcohol, lead and other metals, and drugs), and spinal cord lesions.

Bones and Joints Affections: Rheumatoid arthritis, arthritis deformans and other joint affections, multiple myelitis, Paget's disease, and various bone degenerations and deformities are not uncommon.

Syphilis in the Aged: This may be the result of infection during youth, and may cause a large variety of conditions. It may affect the nervous system (brain, spinal cord and peripheral nerves), the cardiovascular system (causing myocarditis, aortic insufficiency, aortitis, aneurysm and peripheral circulatory disturbance); the gastrointestinal tract (causing gumma of the stomach, liver, and various other diseases of the liver). It may also affect the bones and joints, and practically every tissue of the body.

Premature Senility

Certain pathologic states generally encountered in those past the age of 60, not infrequently occur in younger individuals as the result of disease which causes them to develop senile changes, so that at 30 or 40, such individuals are pathologically old

Progeria (senilism): This is a primary or congenital premature senility of childhood associated with infantilism It is characterized by infantilism, baldness, emaciation, arteriosclerosis, and general decrepitude. Death may occur at an early age from angina pectoris or other senile diseases.

rotic changes may not appear until very late in life.

(*b*) *Intoxicants:* These may be due to disease, lead, arsenic, dietary indiscretions, alcohol (?), and other toxic substances.

(*c*) *Stress and strain:* Insufficient rest and overwork may be factors in intensifying the physiologic sclerosis of the aged to a pathologic degree.

(*d*) *Renal Disease:* Infections and syphilis may hasten or cause arteriosclerotic changes.

In senile arteriosclerosis the *larger arteries* are dilated and tortuous; they are hard, pipestemlike or may be beaded The *aorta* may develop rough calcareous plaques in the intima, or there may be subendothelial softening with the formation of atheromatous ulcers. In the *smaller vessels* the media may undergo calcification and degeneration, the so-called Monckeberg type. Senile arteriosclerosis, by limiting the blood supply of the various organs and tissues, interferes with their functions. It may cause intermittent claudication and other circulatory disturbance. When occlusion of the peripheral vessels occurs, gangrene or trophic ulcers result.

The Heart: Myocarditis with cardiac enlargement may be the result of arteriosclerosis or it may be due to primary affections of the myocardium. Heart disease in the aged may also be the result of rheumatic diseases, emphysema, asthma, renal failure, disease of the liver, hypertension or hypotension, strain and overexertion. The heart is usually enlarged; the rate may be between 60 and 70 per minute, the blood pressure is generally low; occasionally it is high. A loud systolic murmur is usually heard over the entire precordium; this is generally due to sclerosis of the aortic valve, and occasionally to sclerosis of the mitral valve. When the heart is dilated and the valve orifices are also dilated, a diastolic aortic murmur may be heard. A double aortic or double mitral murmur may be due to sclerosis or to rheumatism. *Syphilis is a potent factor.*

The Respiratory System: Chronic bronchitis, bronchiectasis, emphysema and pulmonary fibrosis are fairly common infirmities of old age These may be the result of sinus infections, bronchitis or other bronchopulmonary infections at an earlier age, or they may develop gradually as the tissues lose their elasticity and the blood supply diminishes. Bronchopneumonia and lobar pneumonia are more serious in the aged than in the young, and are often terminal diseases.

The Urogenital System: Among the *kidney affections* of old age are the so-called senile kidney, renal sclerosis or interstitial nephritis The disease runs a chronic course and is usually associated with diffuse arteriosclerosis. It may terminate in uremia or with some vascular accident.

The *prostate* is a most troublesome gland in the majority of old men It may undergo malignant change or there may develop benign hypertrophy Enlargement of the prostate, of whatever cause, produces urinary difficulty and cystitis Prostatectomy is an operation of the aged (SEE p 716). Carcinoma of the *uterus* usually occurs between the ages of 45 and 60, though it may occur at any age (SEE p. 702).

Gastrointestinal System: Carcinoma of the stomach and colon is usually a disease of those between 50 and 65, though it may occur earlier or later Gastric and duodenal ulcers may cause serious trouble when they occur in the

CHAPTER XXXII

Special Examinations—Industrial, Life Insurance, Malingering and Periodic Health Examinations

Industrial Medicine and the Examination of Industrial Workers

Industrial Medicine may be defined as that branch of medical practice which is concerned with the supervision of the general health and the specific problems of preventing disabilities among industrial workers. It differs from the general practice of medicine only in that the worker is selected according to his physical fitness for special jobs, and that hazards peculiar to certain industries are to be prevented or minimized. *Among the 50,000,000 or more workers in the hundreds of industries in this country, there arise numerous problems of how to prevent various industrial diseases* and accidents, and of how to prevent contagion to other workers and the spreading of infection generally The industrial physician is charged with the selection of the physically and at times the mentally fit individual for certain jobs, with the maintenance of health of the workers, and with the treatment of accidental and other injuries so that the efficiency of the worker is not lowered and the industry in which he is employed is not hampered

It is just as important for the industrial managers to choose a properly qualified physician as it is for the industrial physician to choose suitable workers

The Industrial Physician· The Conference Board of Physicians in Industry defined the industrial physician as "one who applies the principles of modern Medicine and Surgery to the industrial worker, sick or well, supplementing the remedial agencies of medicine with the sound application of hygiene, sanitation, and accident prevention." The efficient industrial physician should not only be an alert and competent practitioner of Medicine and Surgery, but should acquire special *knowledge of the hazards of the particular industry in which he serves and the methods of removing them or reducing their danger to a minimum*

Industrial physicians are of two types The part-time worker, and the full-time worker.

Function of the Part-Time Physician: The part-time physician is called upon to examine workers when those in charge think it necessary, and to treat accidents when they occur. His function is that of any practicing physician who is called upon to examine or to treat a patient. An added duty of his is to return the sick or injured to his job as soon as possible.

Function of The Full-time Industrial Physician: His function is threefold ·

I The examination of persons applying for positions Such examination is required for two reasons:

(a) To determine the physical and mental fitness of the applicant to perform the required duties

(b) To weed out those who are physically *unfit for the job but attempt to secure it so that* they may claim "workmen's compensation."

The Place of Geriatrics in Medicine

To prevent the occurrence of many of the diseases of old age or minimize their deleterious effects, it is necessary to prevent or thoroughly cure the preventable or curable diseases of youth, and to teach the young the principles of hygiene and sanitation.

It is also very important for the medical profession to study Geriatrics more intensively. It is a comparatively new field in which too little time has been devoted to comprehensive study Now that the number of old people is increasing study should be devoted towards keeping the aged well and to further increase their usefulness during their lengthened span of life

The neglect of the study of Geriatrics or Senescence may probably be attributed to innate human peculiarities The young man is too busy with active life and old age is to him an unimportant subject; and with the old man it is too personal a subject, or he may lack the initiative to start a study in a new field

The importance of studying the infirmities of the aged with a view of minimizing their helplessness and of increasing their self-respect and their economic usefulness may be gleaned from the fact that the census of 1930 showed that persons aged 65 or over constituted 5 4 per cent of our population Of this group, J. K. Folsom[1] says, "47 per cent are supported in part by relatives; 30 per cent by public assistance, or private charities, and only 33 per cent are self supporting" The preliminary report of the U. S Census of 1940[2] shows that persons 65 years of age and over numbered 8,956,000, an increase of 35 per cent over the number in this age group in 1930 In other words, this group which constituted 5 4 per cent of the population in 1930 increased to about 7 per cent in 1940 It is estimated that the future population of the aged will constitute about 15 per cent of our population. In the words of L K. Frank [3] "We are in process of changing from a large dependent child population to a large dependent aged population," and Christian[4] states: "The changes in quality and proportion of population in various age groups are increasing the importance of Geriatrics at the expense of Pediatrics"

[1] Folsom, J. K Am Jour of Orthopsychiatry, 10 30, 1940

[2] Dept of Commerce, Bureau of Census, Washington, D C, p 5, No 1, 1941

[3] Frank, L K Am Jour of Orthopsychiatry, 10 39 1940

[4] Christian, H A Am. Int Med, 12 1499, 1939

8 Examination of abdomen, genitalia, extremities and prostate in men.
   (a) Hernias.
   (b) Venereal and skin diseases
   (c) Varicosities or flat-foot.

Where the history of the case indicates some abdominal or pelvic trouble in the female employe, a further and more thorough examination in the presence of a nurse or the mother should be made If refused, the person should be sent to the family physician and a report asked for

9 A routine urinalysis in all cases—albumin, sugar and microscopic.

10 Blood-pressure and blood examination in all cases where history and physical examination show they are indicated.

11 Inspection of the teeth of employes by a dentist who recommends treatment when needed, is a valuable adjunct.

12. Wassermann, Kahn or other serologic tests should be made in food handlers, heavy occupations or when syphilis is suspected

Health Defects as Determining Occupation

The defects disclosed should determine whether the person presented should be rejected or accepted as "qualified as to conditions." The aim of such an examination should be

1. Avoidance of injury to the health of the individual inspected
2. Protection of other workers.
3 Maintenance of legality, this directly protects the employer.

Heart Lesions: The heart is examined with the chest bared, and should not be confined to those cases where the physical appearance or a history of previous illness, such as rheumatic fever or syphilis, emphasizes the necessity of such examination When a cardiac lesion is found, the physician must, of course, try to avoid confusion between *functional* and *organic* heart disease, and endeavor to control the data of auscultation by other methods such as percussion, exercise tests, blood pressure reading, and examination of the lungs, liver and extremities for signs of heart failure. In doubtful cases, an electrocardiographic study should be made.

Dearden,[1] an English "industrial surgeon," says, that the main points to be reviewed by the certifying surgeon in considering the bearing of a definite heart lesion on particular employment are:

"1. The already-existing demand for a steady increase in nutritive effort to meet the needs of bodily growth and development.

2 The capability of the organ to answer the call for further increased activity to keep pace with additional tissue change associated with active labor.

3. The power of the organ to resist strain

4. The liability to increased strenuousness of occupation at a later stage.

5. The liability to further attacks of acute rheumatism.

6 The nature and extent of the lesion, and amount of compensation."

As regards the first three headings, certain occupations are of such a strenuous nature from the outset that an imperfect heart could not meet their demands and at the same time supply the ordinary bodily needs. Examples of such types of labor will readily occur to the examiner The fourth heading has to do with occupations where the young person on starting is not put to very hard work, but where, in the course of time, the labor will become more and more arduous The fifth heading has

[1] Dearden. The Medical Examination of the Worker, The Industrial Clinic, edited by E. L. Collis, Wm. Wood & Co.

II. The examination of persons already in industry, for determining their *continued fitness in their occupations* and to detect incipient disease or infection.

III. The supervision of the sanitary conditions and the prevention of avoidable hazards in the place of employment, so as to guard against disease and injury.

I. The Examination of a Person Applying for a Position in Industry

In order to be able to judge properly the fitness of a candidate for a certain *position, the examiner should be familiar* with the type and the various processes of the work that will be required of the applicant, so that, after an examination, he may judge not only the mental and physical fitness of the candidate for such work, but also the length of time (barring accidents) the worker would be fully efficient.

Persons who are required to do laborious work must of necessity have good muscular development, a strong heart, *normal lungs and normally functioning* kidneys. Certain types of laborious work require of the worker, in addition to a strong general physique, sound limbs, normal development of the special senses and a certain amount of skill and judgment. Occupations such as letter carriers, stevedores, soldiers (infantry men) and others who have to walk a great deal must necessarily possess sturdy lower extremities and good feet Those working in chemical industries, gas *works and certain metal trades must* have at least a normal sense of smell so as to detect early the accidental escape of noxious gases. Workers at hazards such as with moving parts of any type of machine, loom, locomotive, crane, buzz saw, lathe, etc., should have quick perceptive powers, and mental and physical ability to act in emergencies to avert catastrophes.

Not all industries require an equal amount of physical fitness, as an instance, cigarmakers, tailors (hand sewers), embroidery workers, bookkeepers, etc, need not necessarily possess perfectly normal *hearts in order to carry on their occupations* successfully Broommakers may be blind; shoemakers may be lame; bakers may be deaf, etc., and still be good and useful mechanics. The requirements of *greatest importance to all types of industry* are that each person employed must be free from contagious and communicable disease, and be mentally and physically fit to do his particular kind of work and at the same time not be a disturbing factor to his co-workers.

Though in this country the law does not require that every individual (except food handlers) before being employed should undergo a *preliminary* physical examination, it is becoming quite common for employers to make a practice of having new "hands" certified by a competent physician as to their fitness to undertake the work proposed.

The following outline is sufficiently detailed and practical for industrial examinations.

1. History of patient on regular blank.
   Personal and family history.
2. Temperature, pulse, weight and height.
3. General inspection—color, nutrition, any deformities or congenital malformations, gait, etc.
4. *Inspection of mouth, teeth, throat.*
5. Inspection of eyes — Snellen test for acuteness of vision.
6. *Inspection and palpation of neck.*
7. Thorough examination of bare chest:
   (*a*) Lungs.
   (*b*) Heart.

exclude all candidates whose lungs, bronchi and accessory sinuses are not in a perfectly normal condition (SEE p 360)

Skin Affections: Though industrial workers, like other members of the community, are likely to be subject to almost any skin disease, according to the hygienic conditions under which they live, and other causes beyond the control of the employer, certain occupations tend to produce special dermatologic lesions. Among young applicants for work it is usual to find impetigo and discrete pustular eruptions, and such parasitic infections as scabies and ringworm, and occasionally pediculosis All such conditions are easily detected and, with the co-operation of the person under examination, readily cured Serious skin lesions usually demand the attention of a specialist

Certain skin lesions may develop upon the hands and face of those working with certain chemicals, dyes, and other substances to which a particular individual may be sensitive or allergic Skin lesions upon the hands may develop among match workers, hatters, x-ray and radium workers and bakers It is of particular importance to exclude from food handling such persons as have skin lesions, contagious diseases, or filthy habits

Deformities: The skeletal deformities most often encountered by the industrial physician are spinal curvatures, tuberculous knee, hip-joint disease of long-standing, the effects of rickets, and, less frequently, infantile paralysis It requires rather keen judgment to decide just how extensive such deformities must be in order to disqualify an individual from engaging in more or less arduous labor Shortness of stature will naturally prevent entrance to a good many occupations, and the general physical examination will have to be relied upon to give information as to how well equipped physically a lame or hunchbacked applicant may be to do the work which he is desirous of undertaking. Not infrequently the affections of childhood have been outgrown sufficiently to permit such an individual to work as efficiently as those who have no skeletal deformities

Eye Diseases and Visual Acuity: Good eyesight, according to Dearden, ranks next in importance to a sound heart for occupational purposes. Defective vision may be due to injury, disease, or errors of refraction, or traumatism may cause cataract, or other forms of injury, which may mean more or less complete blindness of that particular eye, in other cases one eye may have been removed because of injury or disease Defective vision is, however, more often due to errors of refraction than to any other cause; the most common defects of this sort being hypermetropia, mostly unequal and accompanied by marked convergent squint. Though many of these patients will be wearing glasses, there has frequently been failure to properly "educate" the weaker eye, so that it may be wholly useless In making his decision as to the influence a given visual defect will have upon the ability of the worker to carry on his selected occupation, the examiner should take into consideration the following points:

1 The possibility of removing the defect by appropriate glasses.

2 The possibility of injury to the good eye.

3 The extra liability to accident from restricted visual field

4. The liability to eye strain.

5. The possibility, or otherwise, of becoming efficient at the work sought.

The most common eye affections encountered in industrial practice are

especial reference to work in hot and humid atmosphere, wet processes and where there is exposure to weather. The sixth has relation to the condition of the heart itself, and is entirely a matter for the technical judgment of the examiner Where there is evidence of dilatation, particularly when associated with a history of a recent attack of rheumatism, tonsillitis, chorea, etc, or of other acutely serious conditions, the young person is unfitted for any work Arduous labor should not be permitted when there is any definite heart lesion, irrespective of degree. Though in many cases the definite signs of valvular incompetency disappear as the muscular wall regains its proper control over the heart function, with a growing youth the imposition of additional strain will tend to nullify any such tendency. Where there is good compensation, employment of a suitable nature is not barred. There are many light duties which such a young person can perform, even in what, at first thought, appears to be very arduous occupations, and this author concludes by saying that "it is often possible to find work for moderately severe cases, as in an instance of my own where a youth, after several rejections, gravitated to a cork factory, and was 'certified' for sorting bottle corks" If active chorea exists all work about machinery should be prohibited, and if any work is permitted it is a good rule to make an additional point of barring lifting and carrying heavy weights of any kind.

Anemia: Anemia, especially in female employes, should be carefully estimated if found to be present, and its possible effect upon the girl's working capacity considered. If there is a history of vertigo and syncope, work about machinery of any kind should not be permitted, and even if the anemia is of a mild degree, proper treatment should be at once instituted, the condition of undertaking or continuing the work under consideration, being conditional upon reasonably prompt response to therapy.

Respiratory Diseases: Tuberculosis is, of course, the disease which is of greatest concern to the ordinary industrial physician No worker demonstrated to be infected with tubercle bacilli should be permitted to continue at indoor employment, both for his own welfare and that of his fellows. The detection and establishment of a diagnosis of tuberculosis have already been dealt with (See p. 370) so it remains only to offer a word of caution in regard to other respiratory affections which are frequently encountered among industrial workers Where chronic bronchitis is present, the individual should not be permitted to engage in any occupation which will subject him to the constant inhalation of dust, or compel him to remain the greater part of his time in dark or damp working places. If the employee complains of asthma or hay-fever, the proper cutaneous test should be applied, and advice given according to the results obtained. Discharges from the nose, and "catarrh" are most likely to be connected with hypertrophied tonsils and adenoids Such conditions are very common, and while they should not be classed as respiratory diseases, their consideration must usually be taken up at the same time.

Respiratory diseases may develop among coal miners, stone cutters, sand blasters, silica workers, asbestos workers, wool sorters, weavers, bakers, grain and flour workers and bird handlers; also among glass blowers, grinders and gas workers. In these industries it is most important that the examiner should

exclude all candidates whose lungs, bronchi and accessory sinuses are not in a perfectly normal condition (SEE: p. 360)

Skin Affections: Though industrial workers, like other members of the community, are likely to be subject to almost any skin disease, according to the hygienic conditions under which they live, and other causes beyond the control of the employer, certain occupations tend to produce special dermatologic lesions Among young applicants for work it is usual to find impetigo and discrete pustular eruptions, and such parasitic infections as scabies and ringworm, and occasionally pediculosis. All such conditions are easily detected and, with the co-operation of the person under examination, readily cured. Serious skin lesions usually demand the attention of a specialist.

Certain skin lesions may develop upon the hands and face of those working with certain chemicals, dyes, and other substances to which a particular individual may be sensitive or allergic. Skin lesions upon the hands may develop among match workers, hatters, x-ray and radium workers and bakers. It is of particular importance to exclude from food handling such persons as have skin lesions, contagious diseases, or filthy habits

Deformities: The skeletal deformities most often encountered by the industrial physician are spinal curvatures, tuberculous knee, hip-joint disease of long-standing, the effects of rickets, and, less frequently, infantile paralysis. It requires rather keen judgment to decide just how extensive such deformities must be in order to disqualify an individual from engaging in more or less arduous labor. Shortness of stature will naturally prevent entrance to a good many occupations, and the general physical examination will have to be relied upon to give information as to how well equipped physically a lame or hunchbacked applicant may be to do the work which he is desirous of undertaking. Not infrequently the affections of childhood have been outgrown sufficiently to permit such an individual to work as efficiently as those who have no skeletal deformities

Eye Diseases and Visual Acuity: Good eyesight, according to Dearden, ranks next in importance to a sound heart for occupational purposes Defective vision may be due to injury, disease, or errors of refraction, or traumatism may cause cataract, or other forms of injury, which may mean more or less complete blindness of that particular eye, in other cases one eye may have been removed because of injury or disease. Defective vision is, however, more often due to errors of refraction than to any other cause; the most common defects of this sort being hypermetropia, mostly unequal and accompanied by marked convergent squint. Though many of these patients will be wearing glasses, there has frequently been failure to properly "educate" the weaker eye, so that it may be wholly useless. In making his decision as to the influence a given visual defect will have upon the ability of the worker to carry on his selected occupation, the examiner should take into consideration the following points:

1. The possibility of removing the defect by appropriate glasses.
2. The possibility of injury to the good eye.
3. The extra liability to accident from restricted visual field.
4. The liability to eye strain.
5. The possibility, or otherwise, of becoming efficient at the work sought.

The most common eye affections encountered in industrial practice are

Medical Examination for Life Insurance

When a physician accepts an appointment as a medical examiner for an insurance company he becomes in fact an employee of that company, and his acceptance of the appointment implies his willingness to serve the best interests of that company and to be worthy of the confidence which it has imposed in him. If he feels that this is an "unprofessional" attitude, and that he cannot put the interests of a corporation before those of the individual patient in any transaction, then he will do well to leave insurance examination to someone else. As a general rule the demands of insurance examinations do not in any way conflict with the most scrupulous requirements of medical ethics. The best interests of the insurance company are always served by an observance of the strict adherence to exact statements, and painstaking thoroughness in examination and diagnosis. To be a successful medical examiner one should have a most thorough training and experience in physical examination, but though nothing can take the place of practice in this, as in most other branches of medicine, a few suggestions may be helpful to those who are undertaking life insurance examination for the first time.

The suggestions which Henry Wireman Cook of Minnesota set forth some years ago are so pertinent and well expressed that the liberty is here taken of borrowing rather freely from his remarks.[1]

[1] Cook: Suggestions to Medical Examiners for Life Insurance. Journal-Lancet, 32: 281, 1912.

Beginning with little more than inspection of an applicant by the examiner, companies have gradually come to expect a fairly complete clinical history, and a thorough physical examination, supplemented by the recommendation that the applicant shall or shall not secure the policy for which he has applied. This requirement demands a knowledge of history-taking, the ability and practice necessary for a thorough physical examination, and in addition, sufficient general knowledge of insurance data to correlate the history of the case and the physical examination with the habits, occupations, social status, financial standing and insurable interest, so that an intelligent prognostic opinion may be given.

Most of the questions to be asked by the examiner of the applicant are explicit and need no elaboration. The need for the examiner to furnish all explanations in regard to every illness or impairment cannot be too strongly emphasized, as it is the most frequent cause of unnecessary correspondence and annoying delays. If a man says he was treated by Dr. X for "biliousness" or "indigestion" six months ago, a full explanation is absolutely necessary, with a statement, if obtainable, from the attending physician. It is well known that laymen speak vaguely of "indigestion," "cold," "biliousness," etc., and offer them as a satisfactory description for any number of conditions, varying from trivial constipation or coryza, to gall-stones, advanced phthisis, or even carcinoma. It is obviously impossible to accept an applicant's diagnosis without inquiry as to the symptoms and

the course of the disease, or without a statement from the attending physician. The necessity for these details is stated in every examination blank.

Form of Questionnaire

Each company has its own forms in which the information obtained is to be set down by the medical examiner, but whatever the form the general trend of the questionnaire will be found to be the same in practically all cases. It will always be necessary to obtain the following information:

I Objective Examination of Applicant:
- (a) Identification
- (b) Age
- (c) Occupation
- (d) Race.
- (e) Sex.
- (f) Marital state

II General External Examination
- (a) General appearance.
- (b) Detailed appearance of head, trunk and limbs
- (c) Posture and gait.
- (d) Physical defects and deformities
- (e) Stigmata of degeneracy

III History
- (a) Family history
- (b) Personal history
- (c) Habits
- (d) Associations and amusements (those bearing on health)

IV Physical Examination:
- (a) Chest—lungs
- (b) *Heart and blood-vessels*
- (c) Abdomen.
- (d) Urinalysis, with other laboratory tests as required by the company, or deemed needful by the examiner.

I. Objective Examination

Identification: It is essential that the medical examiner should be certain that the individual he is examining is the same person for whom application for life insurance has been made. Though insurance frauds are not now as "fashionable" as they were a few years ago, the attempt to induce the medical examiner to send in a favorable report may even extend to the substitution of a better "risk" than the one actually applying for protection, so in any case where the physician is not personally acquainted with the applicant before he becomes the subject of examination, the physician must make sure that there has been no mistake in the identity. If a personal introduction from the agent is not possible, he can, for instance, question the applicant on the part of the application that was completed by the agent and the answers should correspond to the recorded data as to the time and place of birth, name and residence of beneficiary and other insurance carried, if any. Preliminary to the examination, the examiner may also ask for the applicant's signature, and compare it with the signature secured by the agent on the face of the application; or he may secure from the agent an accurate description of the person to be examined so that he cannot possibly make an error in examining any other than the proper person As it sometimes becomes necessary to identify the holders of insurance policies after death, it is desirable to note carefully the location, size or other characteristics of any moles, scars, nevi, or other marks and deformities which might aid in the identification The condition of the teeth, the presence of artificial dentures and fillings may also be of help in post mortem identification, it is therefore wise to be prepared for this emergency by obtaining identification data at the very outset

Age: As it is upon the basis of age that all life insurance premiums are de-

termined, and all companies have definite age limits within which they accept risks, the exact age of each applicant must be *determined beyond question. If there appears to be any reason, no matter how trivial, to lead the examiner to believe that the applicant has made a false statement in regard to this point, he must make it clear that the possibility of obtaining the desired policy rests wholly upon the establishment of this fact.*

If the applicant looks older than the stated age, the physical examination may quickly disclose the reason for the apparent discrepancy; indeed, the personal history is very apt to give the clue even before the examination is begun. In the words of Ramsey: "It may be due to over-work or strain, dissipation, some external agency, business worry, a deep-seated disease, or *the culmination of a process beginning in youth and finding expression in middle age The family history and the environment of his progenitors have much to do with apparent age; if a man's parents have tuberculosis in any of its forms, or have through poverty or unfortunate business associations* been compelled to undergo hardships, it necessarily has its effect upon the offspring. The same effects are seen in those born of neurotic parents; their resistance is lessened to diseases of childhood, of which the marks are left; their vitality is lowered, the results being the expenditure of more energy in coping with business propositions and inability to endure prolonged physical exertion, thus causing them to become *prematurely old.*"

Though the calculation of the expectation of life in any given case does not come under the points which the medical examiner *must cover in the issue of a policy, it is well for him to be able to compute it for his own satisfaction.* The rule for determining it by the Actuaries' or Combined Experience Table is as follows: If the applicant is 40 years of age or older subtract the actual age from 80 and divide the result by two-thirds, the result will give the expectancy of life; if the applicant is between 20 and 40 years of age add one year to the result obtained as above

Occupation: Extra-hazardous occupations are usually designated by each company in their directions to their medical examiners. Most states now have some laws regulating those trades and vocations which are rated as injurious to health and tending to risk or shorten life. The necessity of obtaining details as to the applicant's occupation, and correlating it to the history and examination findings is very obvious. *There is also what is known as the "moral hazard" which occurs in occupations which tend to expose those who follow them to dissipation and excesses of various kinds. Thus, other things being equal, a clergyman is regarded a better risk than an actor, and a milkman than a bartender of like age and physique "The influence of occupation on longevity has been carefully computed, as applying to general principles, but the examiner must judge in individual cases from what has been elicited by the examination whether the applicant's vocation is compatible with long life, i e, whether his physical powers and ancestral history are sufficiently good to neutralize any prejudicial influence occupation may have on his life." According to most authorities on life insurance examination, players of wind instruments are likely to be sub-*

ject to emphysema, varicose veins are found in shop-clerks, motormen, policemen, and those who are required to assume the standing or semi-erect posture for long periods of time; lead and other poisoning are apt to be prevalent among painters, gilders and those engaged in the manufacture of articles used in these and allied trades compelling them to come in contact with poisons; bronchitis and phthisis occurs rather frequently in grinders and polishers of metals, marble cutters and printers. Those vocations requiring exposure to the inhalation of irritating vapors, toxic substances and all kinds of effluvia predispose to laryngeal troubles and may invite lung infection

Race, Sex and Marital State: The susceptibility of particular races to certain diseases is common knowledge in medical practice Lister,[1] an Englishman, giving advice to his medical compatriots on the practice of life insurance examination remarks that, "as every race in the world is insured by English offices 'every racial peculiarity is, therefore, a matter of importance to the insurance examiner,' and in the United States, the 'melting-pot of all nations, this is even more true' It is often said that because many Germans are obese, an obese German is not to be regarded necessarily as bad a life insurance risk as an obese Englishman A large abdomen is also common among Belgians, French, the Dutch, Italians and Spaniards" To English examiners the question of change of residence from the mother country to the tropics or *vice versa*, is a more urgent one than with us, but nearly every insurance examiner will occasionally find cases where such circumstances must be considered. A history of previous residence in a tropical climate should put one on his guard for diseases which may be latent in the applicant's system, and may recur, such as chronic dysentery, tropical sprue or malaria

Formerly, it was an exceptional thing for any company to insure the lives of women, but the practice is now common Outside the child-bearing period, the cold, unsentimental evidence of statistics shows that female lives are better risks than male lives No company intentionally issues a policy on the life of a woman known to be immoral, and those with an antecedent history of gonorrhea or syphilis can seldom secure life insurance The medical examiner will usually find his company slow to accept his recommendation of the mother of an illegitimate child, though from the physical standpoint she may be an excellent risk The question of insuring the lives of women who have undergone pelvic operations has been much discussed by physicians interested in insurance examination, and there appears to be a wide diversity of opinion about it A woman past the menopause is generally conceded to be a better risk than a man of the same age

Generally speaking, married people are better risks than single ones Their habits of life are apt to be more settled and regular, and proper exercise of the sexual functions tends to prolong life Single women over thirty are better risks than single men of the same age, and for each year thereafter the woman becomes a better risk, and the man a poorer one Marriage late in life tends greatly to increase the risk, especially if there is great disparity in the ages of the parties

[1] Lister Medical Examination for Life Insurance London E Arnold 1921

II. General External Examination

General external examination is conducted along the same lines as in any branch of medical practice. As the time allotted to make these examinations is usually quite limited, the examiner will be obliged to depend more on the general impression made upon him by the applicant's outward appearance than is usually the case in private practice Bearing in mind the conditions which are most likely to render an applicant uninsurable, the examiner should look for indications of their presence as soon as the applicant appears. The expression and color of the face may suggest the possibility of anemia, nephritis or tuberculosis, or of addictions such as alcohol and narcotics; and the posture and general "build" may be indicative of the general condition. A "good risk" will usually stand erect and have an air of strength and buoyancy, while disturbances in the nervous system and the physical characteristics of degeneracy can often be correctly surmised even when the gait and position are but slightly abnormal.

III. History

The elicitation of the *personal and family history* must follow the lines laid down in the examination sheet, but frequently the medical examiner will be called upon to use his judgment in interpreting the findings. One point frequently overlooked is a change in the habits and manner of living at or near middle age. It is a very common thing for men who have risen from poverty to the possession of a competence or affluence to apply for a policy when "things are easier" for them. It is sometimes a difficult question to decide just what effect these changes will have upon an individual's longevity. In a study of gall-stone disease, one writer remarks that a change "from walking to a buggy which one drives himself is one thing, and a change from walking to motor car and hired chauffeur quite another." The "moral hazard" here must also be carefully considered with both men and women.

The examination blank always devotes considerable space to the family history, and every examiner knows how difficult it is to fill in these questions satisfactorily. Frequently, the applicant will have surprisingly little knowledge of the medical history of even his nearest relatives. Many are unable to tell the age at death of their grandparents, or even of their parents The cause of deaths which have occurred even within their immediate knowledge will be practically unknown to them, or will be attributed to "stomach trouble," "heart disease," or something else equally vague. All the examiner can do when such knowledge is vague, is to question as minutely as possible regarding symptoms, duration of the illness or other circumstances likely to give evidence as to exact cause of death or the ailments which preceded death Lister points out that "death from 'pneumonia, bronchitis or pleurisy often covers tuberculosis.' . . . A mother's or sister's death is often ascribed to 'childbirth' or a 'confinement'," when actually "a confinement given as a cause of death may be only a phase in which the family has been instructed and one which is used to cover phthisis. The death may have occurred some months after the child was born, and the death certificate will shed a different light on the matter."

Heredity in all its phases has a very important bearing on the issuance of life insurance. "Old age" is often given as a cause of death, but even where the parents have attained great ages it is wise to obtain exact data, so far as possible. As a contrast to old age in the parents there is what Lister terms the "early breakdown age" The examiner will often be told that the applicant's parents and perhaps several brothers have died before fifty or sixty of various diseases, such as bronchopneumonia (so-called), pernicious anemia, cancer, typhoid fever and Bright's disease, and if these deaths all occurred between forty-five and sixty, even if the applicant is in apparent good health, such a family history strongly suggests "deficient vitality"

"General paralysis of the insane" in a parent is often given as simply *paralysis* If the applicant states that "paralysis" was the cause of a relative's death, the exact kind of paralysis should be ascertained If the death of a parent were due to dementia paralytica it is important to find out the age of the applicant at the time the parent died, and also to keep watch for the stigmata of syphilis in making the physical examination Insanity and epilepsy are always serious factors in a family history Even a history of "accidents" may turn out not to be so "accidental" after all Lister suggests that "such histories may also conceal suicide as a hereditary tendency, a point to be remembered."

Concerning *habits, associations* and *amusements* something has already been said under previous headings. The danger of contracting pulmonary tuberculosis, *for instance, from infected fellow* employees or housemates, is just as great as *when this disease is actively* present in a member of the applicant's immediate family. Similarly, a man who spends his hours of leisure in pool-rooms and crowded "shows" is not likely to prove as good a risk as he who finds his recreation in golf or tramping, or even in quietly reading at home. Taken by themselves such points are of trifling value, but considered in conjunction with the physical findings, the purpose for which life insurance protection is desired, and other matters with which the medical examiner has to do, they are often of profound significance.

IV. Physical Examination

The *physical examination* proper may be divided into four essential parts While for the meagre fee given by most insurance companies and the short time usually allotted to making the examination, even an acute diagnostician could not always be expected to exclude such conditions as leukemia, carcinoma of the rectum, tuberculosis enteritis, and many other of the less common diseases, it is not unreasonable to expect that an applicant who has been unreservedly recommended by the physician who examined him should have the general appearance of average good health, that his lungs should show no evidence of active disease, that the cardiovascular system should be approximately normal in structure and function, and that the kidneys are not excreting albumin and sugar These are practically the only requirements of the insurance examination, except on some blanks, temperature and reflexes are also questioned, yet the number of applicants with pulmonary tuberculosis, cardiovascular disease and nephritis who annually "get by" the average medical examiner will

be evident to anyone who examines the "paid claims" of any company, and notes how many who have but recently taken out policies have succumbed to those diseases.

General Appearance: An unhealthy or under-standard general appearance is frequently neglected by an examiner. He is intent upon the discovery of definite signs of disease, and his negative physical findings are apt to offset unduly the general impression which he may have formed at first sight; or he may have entirely overlooked the pallor of an anemia, the slight cyanosis of faulty circulation, the sallowness of interstitial nephritis, the wrinkles from recent loss of weight, etc An applicant who looks sick or frail should not be recommended as a first-class risk merely because the examination fails to reveal any definite signs of disease

Pulmonary Examination: The examination of the lungs is perhaps the most unsatisfactory portion of the average examiner's report. The cause of this is twofold. First, evidence of pulmonary disease is often vague; and sometimes, even in advanced stages, entirely lacking to all but the lung specialist; secondly, the interpretation of the pulmonary findings demands more skill and experience than is required by any other physical signs When one realizes that arrested cases of even advanced phthisis may reveal no adventitious sounds nor any change in percussion resonance, when the lungs in a recent case of hemoptysis may appear normal, and when a suspected case of incipient phthisis may undergo repeated clinical examination before any abnormality is positively identified, it is evident that there is an excellent excuse for the recommendation of a certain proportion of tuberculosis applicants But with all due latitude for the difficulties of the latent and incipient cases, the large proportion of tuberculous applicants accepted each year can be accounted for only by carelessness or inexperience on the part of the examiner Failure to bare the chest of all clothing, a step so essential in every physical examination, is accountable for some of the cases overlooked. A quiet room and a proper light are also necessary Often proper inspection guides our attention to the diseased areas

In the pulmonary examination there should be percussion over at least seven different areas on each side, comparing the two sides step by step above and below the clavicle, and at the second intercostal space in front, in the axillæ, and in three areas in the back One of the best guides to involvement of the apices is to have the applicant breathe deeply during inspection and palpation, and to note if the apices become resonant on percussion above the clavicle, thus noting whether the lung moves freely, or appears restricted by pleural adhesions. Delayed expansion over a restricted area or over one side of the chest may be detected by inspection, but is best determined by palpation. Localized diminished expansion should put the examiner on his guard; that area should be examined with the greatest of care. Mensuration for determining chest expansion is a valuable aid. The tape measure should encircle the chest in the region of the third rib The circumference of the chest is noted during quiet breathing, then during the deepest expiration, and again during full inspiration. The distance between deep expiration and full inspiration is considered the chest expan-

sion. An expansion of less than two inches is pathologic. The evidences of inflammatory changes at an apex should be sought for in every case. Careful auscultation over the same areas, at least through one whole phase of respiration, should follow percussion, and particular attention should be paid to the detection of râles after coughing or the prolongation of expiration. This procedure should not take over five minutes, and is certainly a reasonable requirement before an examiner should be willing to pronounce the lungs negative A suggestive personal or family history, an elevation of temperature, a respiration rate above 20, should lead to a more minute detailed examination

Cardiac Examination: The cardiovascular examination is second in importance only to the urinalysis, as a single guide to insurability. It is far easier in its essentials than a pulmonary examination and far more definite in its indications. A thickened artery, a heart murmur, a displaced apex and increased area of cardiac dullness and an abnormal blood-pressure are some of the most definite clinical signs and should be positively excluded in every insurance examination

Estimation of the pulse rate should begin the examination, 60 to 90 are the usually accepted normal limits A pulse-rate below 60 suggests increased resistance, high blood-pressure and cardiac hypertrophy, above 90, after nervousness is eliminated as far as possible, one would look for evidence of hyperthyroidism or some cardiovascular weakness. After counting the rate, the examiner should gauge the vascular tension using two fingers on the radial, and estimating the thickness and hardness of the artery after obliteration of the blood stream. The tactile impression of pulse-tension obtained in this way, should be confirmed by the use of the sphygmomanometer. It should be remembered that a thickening and sclerosis of the radial is apt to reflect a similar degenerative change in the cerebral vessels, aorta, heart, and kidneys.

In the cardiac examination, the physician should face the applicant who, in turn, faces a bright light. He should look for any abnormal pulsation, any bulging, especially over the precordium, and any dilated veins The apex beat should be definitely located, displacement to the left being particularly important The visible or palpable apex should be confirmed by auscultation, and murmurs attentively listened for at the apex, second right costal cartilage, and down the left border of the sternum. Cardio-respiratory and functional heart murmurs should be differentiated from organic murmurs. It is interesting to note in this connection that the experience of one company has been as unfavorable to "functional" heart murmurs as to "organic." There are undoubtedly "functional" murmurs which might be negligible in an insurance examination, but the above experience shows that the tendency is to give the applicant the benefit of the doubt in a doubtful case. As attending physicians we are too apt to associate organic heart-murmurs with some sign or symptom of incompetence; and as examiners, we are too prone to call "functional" a soft murmur at the apex, without appreciable hypertrophy, in a robust, active young individual. If a murmur of any kind is found, it should be fully noted, and the notation supplemented by the opinion of the ex-

aminer as to the kind of murmur or its importance. An unduly accentuated pulmonic or aortic second sound or a reduplication of the first or second sound should be faithfully reported. No cardiac examination should be considered complete without a definite attempt to determine the left border of relative cardiac dullness. Where the dull note extends beyond normal limits this fact should be definitely stated Increased area of cardiac dullness, showing either hypertrophy or dilatation, is associated with so many serious conditions that its recognition is of paramount importance.

What is more important than the finding of a cardiac murmur or cardiac enlargement is *the determination of the functional capacity of the heart muscle.* A person of sedentary habits and light occupation who has a mitral regurgitant murmur which is fully compensated may be a good risk, while another person who has no murmur but has a poor myocardium may be a very poor risk. Therefore, every cardiac examination should include an "exercise test" whereby myocardial efficiency may be measured. A simple test is to have the examinee hop 25 to 50 times on one foot or have him walk up and down a flight of ordinary stairs, the rapidity of the pulse should be noted before the exercise, immediately after and again two minutes later In the normal heart the pulse rate should come down to normal within two minutes after the exercise. A fall in blood-pressure or only a slight elevation or a very marked elevation in the blood-pressure after exertion also denotes myocardial weakness (For cardiac Function Tests, see pp. 442 to 447.)

Examination of the abdomen and the lower extremities: These are often neglected in the examination of those who are being insured even for a moderately large policy. This is an inexcusable omission, as there are many diseased conditions which may only be detected by an examination of these parts.

Examination of the abdomen should include:

Inspection as to general contour, size and shape, rashes, scars, and enlarged veins

Palpation is most important for the detection of tumors, large liver, spleen and kidneys, also for the purpose of eliciting tenderness and muscular rigidity over the appendiceal, gall-bladder or other important regions of the abdomen (SEE· p. 586)

Examination should also be made for *hernia.* In the male, particularly if over 50 years of age, the *prostate gland* should be examined

The *extremities* are to be examined for large glands, venous distention, arterial pulsations, ulcers and scars.

Neurologic Investigation: This is also of sufficient importance to require an examination of some of the reflexes, station, gait, etc.

Urinalysis: The most important single feature of the insurance examination report is the urinalysis. By this means the average examiner can detect a condition of impaired health more frequently and more definitely than by any other portion of the examination A discoverable abnormality shown by the urinalysis is probably associated with other conditions of impaired health Excluding diabetes and primary parenchymatous nephritis, which in themselves constitute a large group, the urinalysis

may reveal the low specific gravity of diabetes insipidus and interstitial nephritis; the bloody urine of pernicious malaria, renal and vesical calculus, tuberculosis and carcinoma of the genitourinary tract; the decomposed urine of hypertrophied prostate and cystitis; the pus of an inflammatory condition of the tract; and lastly, and most important, the albuminuria of febrile conditions, of chronic pus absorption, of arteriosclerosis, or any serious toxemia, *i.e.*, chronic tuberculosis, alcoholism, etc In fact, there are few serious diseases not associated with the excretion of at least a trace of albumin and the effect of any of these agents in producing albumin is increased with advancing years as the structural changes of age take place. On the other hand, the examiner should properly evaluate the presence of albuminuria, and bear in mind that a trace of albumin minus casts in an otherwise normal urine may be of no pathologic significance The inability to find albumin because a white ring does not promptly appear when the examiner pours some urine over nitric acid, or if no white cloud shows in the boiled urine after adding dilute acetic acid, may be due to faulty methods of observation as he may look for the cloud from where he stands beside the table or sink at the back of the room without properly shading the test tube, thereby failing to see a faint ring or cloud. Unless albumin is present in large amounts, such methods will not reveal its presence.

Résumé: To put it tersely, it must be stated that the value of a physical examination for life insurance is in direct proportion to the thoroughness and completeness of the examination. The excuse for incomplete and hurried examination is usually inadequate compensation; if that be true then life insurance companies get only what they pay for, or very much less, a circumstance to be deplored because it is unsatisfactory to all concerned. Often a bad risk gets by while a very good risk, because of a minor ailment, is being excluded

Malingering

Malingering is usually defined as the simulation of injury or disease where no pathologic condition is present. Generally speaking there are *two* groups of malingerers: (1) In which a well person attempts to simulate illness; and (2) In which an ill or defective individual tries to hide his illness or defect and attempts to pass himself off as a healthy person.

(1) The malingerers of group one are persons who simulate illness for a selfish motive usually for personal gain such as claiming or exaggerating injury because of an accident, so as to recover damages; feigning sickness in order to collect sick benefits; as excuse for not appearing in court, or as an attempt to evade military service or other duties. Illness or injury may often be feigned by children and adults who are desirous of eliciting sympathy In this connection, it should be borne in mind that the neurotic individual who has a multiplicity of complaints about which he "hollers loud and long" without any apparent cause, is not necessarily a malingerer and that he may have a definite basis for his complaints, only that the loudness and length of his "hollering"

is out of proportion to the severity of *the injury.* This fact is quite often *overlooked and the complaining* person is *styled* a *"neuro" or a malingerer* On the other hand, hysterical or neurasthenic persons may, because of being in contact with certain sick people, or because of the perversity of their nervous systems, attempt to exaggerate their symptoms or mimic disease consciously or unconsciously

(2) Malingerers of the second group are persons who simulate good health *in order to pass life insurance examinations, industrial examinations, or any* examination which would bar *persons* not in perfect health. Such malingering may be found among persons who attempt to show bravery, or are ashamed to admit any deficiency or disease that they may possess. However, the largest number of malingerers of group two in attempting to deny illness or injury are persons who seek life insurance, sick benefit insurance, industrial positions or admission to the federal or *other services* which *pay their sick and injured (if in line of duty)* a *certain weekly or monthly* allowance

It is therefore of equal importance for the examiner to be able to detect the sick person who plays off as well and the well one who plays off sick

It is often a difficult matter to separate hysteria from malingering; often, except for the skilled psychiatrist, it is impossible. Therefore the examiner should familiarize himself *with those* symptoms of *hysteria which cannot be simulated, such as anomalies of secretion,* unilateral hyperidrosis, salivation, *oliguria*, or, very rarely, anuria without uremic symptoms, in order not to be led astray by the malingerer.

The demeanor and behavior of the hysterical subject, according to Jones and *Llewellyn* [1] usually give indications of his abnormal mentality. "The *excitability and restlessness, the hurried extravagant speech, the odd* gesticulations, the *rapid* muscular tremors, the widely-opened eyes, as if frightened, all combine to give a somewhat characteristic expression."

While the hysteric is usually unconscious of the unreality of his symptoms, the malingerer, though his outward appearance may belie him, is often sullen, obviously suspicious, and *ill* at ease. *Though he may protest his good* faith *most volubly, he is careful to* choose his words and avoid conversational pitfalls, while the hysteric subject is so anxious to make a histrionic effect and impress the examiner with the gravity and intensity of his sufferings that he takes no thought of consistency "The hysterical person revels in *examination; it cannot be too long or too minute; the more spectators, the* better he is pleased; he has no rooted objection to being hospitalized, and for medicine and treatment he is a glutton"

The malingerer, on the other hand, "loathes examination, and if he can put off the evil day, he will. *During its progress he is often unconsciously* wilful or sulky, and if he can fasten on to something harsh in the methods or tests employed, he is quick to take umbrage. He often shrinks from treatment, or shirks it wholly, and the certificate once gained, cannot see the doctor *too seldom, and to hospital he will not* go if *he can help it*"

It must be remembered, that most individuals suffering from hysteria dis-

[1] Jones and Llewellyn. Malingering, Wm Heinemann, London.

play bodily stigmata which a careful physical examination with tests of sensation and reflexes will reveal, and their presence supplies data on which a diagnosis may be based, while the absence of any such signs is strongly in favor of malingering.

The chief complaints of the malingerer are subjective signs, only occasionally, when conditions demand it or when instructed by an unscrupulous person, will the malingerer attempt to manifest objective signs The most common subjective signs complained of are pain, vertigo, insomnia, disturbing night dreams, disturbed vision, deafness, anorexia, indigestion, fatigue (mental and physical) and various phases of sympathetic nervous disturbances.

The objective signs most frequently complained of are difficulty or inability to walk, bend or perform any movements Certain objective signs may even be brought out by the use of drugs or other agents Among these may be noted large doses of strychnia and belladonna to cause cardiac palpitation and exaggerated reflexes, various rubefacients to cause local redness so as to simulate inflammation Irritants in the eyes may simulate iritis or corneal disease Rupturing the ear drums with an instrument in order to simulate spontaneous rupture Soapsuds exuding from the mouth during a feigned convulsion may simulate epilepsy. Often pre-existing conditions may be attributed to an injury, as for instance, hernia, uterine prolapse, abortion, pulmonary tuberculosis, pleurisy, cardiac disease, visceroptosis, spinal cord disease, bone or joint diseases, etc. The examiner must, therefore, be on guard not only to differentiate between conditions that may have resulted from the injury and conditions that existed prior to the injury, but he should be able to determine to what degree a pre-existing condition became aggravated because of the injury.

A complete history skillfully elicited and a most careful examination may reveal to the experienced examiner the presence or absence of objective signs and thus he may be able to detect the malingerer, the exaggerator and the honest person. It must be borne in mind that not all persons claiming sick benefits or compensation for injuries are malingerers, as a matter of fact, the majority of claims are just, a small proportion of claimants are absolutely fakers and a goodly number are exaggerators

The confirmation of subjective signs is often difficult or impossible because the examiner cannot disprove the statement made by a patient as to headache, vertigo, buzzing in the ears, disturbed vision, etc. A headache can be felt only by the sufferer; its very existence or severity is entirely a personal matter for which we have only the patient's assurance of its existence. The examiner cannot disprove the existence of a headache, nor its severity The same holds true of vertigo and of many other subjective symptoms. However, a careful history, taken possibly on two or three occasions, close observation of the patient at various times together with a complete physical and whenever necessary, certain laboratory examinations, may often help to establish a correct diagnosis

Simulated Pains and Disabilities

Pain in the back or in the legs alleged to be due to some traumatic acci-

is out of proportion to the severity of the injury. This fact is quite often overlooked and the complaining person is styled a "neuro" or a malingerer. On the other hand, hysterical or neurasthenic persons may, because of being in contact with certain sick people, or because of the perversity of their nervous systems, attempt to exaggerate their symptoms or mimic disease consciously or unconsciously.

(2) Malingerers of the second group are persons who simulate good health in order to pass life insurance examinations, industrial examinations, or any examination which would bar persons not in perfect health. Such malingering may be found among persons who attempt to show bravery, or are ashamed to admit any deficiency or disease that they may possess. However, the largest number of malingerers of group two in attempting to deny illness or injury are persons who seek life insurance, sick benefit insurance, industrial positions or admission to the federal or other services which pay their sick and injured (if in line of duty) a certain weekly or monthly allowance

It is therefore of equal importance for the examiner to be able to detect the sick person who plays off as well and the well one who plays off sick

It is often a difficult matter to separate hysteria from malingering; often, except for the skilled psychiatrist, it is impossible. Therefore the examiner should familiarize himself with those symptoms of hysteria which cannot be simulated, such as anomalies of secretion, unilateral hyperidrosis, salivation, oliguria, or, very rarely, anuria without uremic symptoms, in order not to be led astray by the malingerer.

The demeanor and behavior of the hysterical subject, according to Jones and Llewellyn [1] usually give indications of his abnormal mentality. "The excitability and restlessness, the hurried extravagant speech, the odd gesticulations, the rapid muscular tremors, the *widely-opened eyes, as if frightened*, all combine to give a somewhat characteristic expression."

While the hysteric is usually unconscious of the unreality of his symptoms, the malingerer, though his outward appearance may belie him, is often sullen, obviously suspicious, and ill at ease. Though he may protest his good faith most volubly, he is careful to choose his words and avoid conversational pitfalls, while the hysteric subject is so anxious to make a histrionic effect and impress the examiner with the gravity and intensity of his sufferings that he takes no thought of consistency. "The hysterical person revels in examination, it cannot be too long or too minute; the more spectators, the better he is pleased; he has no rooted objection to being hospitalized, and for medicine and treatment he is a glutton."

The malingerer, on the other hand, "loathes examination, and if he can put *off the evil day, he will* During its progress he is often unconsciously wilful or sulky, and if he can fasten on to something harsh in the methods or tests employed, he is quick to take umbrage. He often shrinks from treatment, or shirks it wholly, and the certificate once gained, cannot see the doctor too seldom, and to hospital he will not go if he can help it."

It must be remembered, that most individuals suffering from hysteria dis-

[1] Jones and Llewellyn. Malingering. Wm Heinemann, London.

leged to be affected, and asks that the knee be raised against resistance The subject, *on attempting to do this, may* say that it causes pain The examiner should then place his hand *beneath* the thigh, and tell him to depress the knee against resistance If he now says that *it causes him no pain, he is probably* malingering, because in the second movement, the lumbar muscles do participate in the movement, so that if the case were genuine the pain would be present or even aggravated, *while in* the first movement there would be no pain occasioned because the movement involves no strain on the lumbar muscles

Again, the malingerer, being unaware of the fact that often different movements are subserved by the same muscle, may be betrayed into contradictions Thus, suppose that the circumstances *of the casualty and the results of the* examination suggest that the lesion is in the latissimus dorsi muscle. The malingerer now, though he protests that owing to the pain he is unable to stand erect, nevertheless, when asked to depress and at the same time carry his raised arm backward, does so without any complaint, unaware of the fact that in both these procedures the latissimus dorsi is concerned Similarly, he may plead pain on rising from a stooping posture, *though conscious of no disability* in standing or walking If a malingerer is told to bend forward and try to touch his toes, he may feign complete rigidity of the trunk and leaning from the ankle joints take the pose of a man preparing to dive, hoping that by suppressing the natural automatic adjustment of the body to the forward-bending position, he may convince the examiner of the impossibility of executing the movements of trunk flexion. Yet, even if the lumbar column be stiff, flex*ion at the level of the hip-joints is still* possible. The examiner should now place the subject upon his back, and ascertain if his hip-joints can be passively flexed If not, it is evident that *the subject is, at least, exaggerating his* disability, for even if the lumbar spine be actually stiff the fixation of the hip joints is voluntary and intentional.

To ascertain the mobility of the in*dividual lumbar segments, the fingers* of both the examiner's hands should be inserted between the spinous processes of the different lumbar vertebræ, and the patient asked to stoop, and then raise himself *slowly* to the erect position. In the normal column, the projecting spines will be found to separate on forward flexion, while they approximate to each other during extension; *and if this can be executed freely and* painlessly there is probably nothing in the nature of structural damage at the site indicated as being painful and tender.

Leg Injury: *When the leg is the* site of pain alleged to be due to trauma, a careful examination of the entire extremity must be made to rule out all genuine pathologic conditions Special sources of fallacy which lead to unfair accusations of malingering are affec*tions of the sciatic nerve, osteo-arthritis* of the hip, sacro-iliac strain, subgluteal bursitis, intermittent claudication, varicose veins, and the local or referred pain of flat-foot, already mentioned in *considering lumbar pain Jones and* Llewellyn consider the most valid evidences of the reality of pain in the leg to be based on the pupillary, sphygmographic and sphygmomanometric reactions, obtained after the directions of

dent is a *form of malingering* frequently encountered in compensation claim work.

Back Injury: In genuine cases of back injury the lumbar column and pelvis are held motionless as far as possible, as the patient endeavors to keep them in the posture which gives him the least discomfort. The following points to be noted are suggested by Jones and Llewellyn:

1. *Is there an absence of the unconscious* normal swaying of the trunk for balance, viz., *rigidity?*

2. Does he bend forward with a list to one or other side?

3. Is the work cautious and groping?

4 Does he sit down gingerly, and when rising place his hands on his thighs, finding support at successively higher levels until he stands upright?

All these or similar automatic defensive adaptations point to fraud, or at least to exaggeration. When asked to strip the examiner should have a sharp eye for any wincing when movements calculated to evoke pain are performed Sometimes, if his trousers slip to the *floor and the examiner turns away* and then asks the man to approach nearer, he may, before starting, stoop swiftly to *raise them to keep his feet from becoming* entangled. Or, when seated upright in a chair he may, when asked to straighten *his knees, do so without* wincing, although this involves strain on the dorsal muscles and fasciæ.

The past history, both of anything in the medical record which might suggest constitutional causes and also of the accident to which the pain is attributed, should be carefully studied. In genuine traumatic cases the onset of the pain is immediate, its intensity straightway excruciating. If the onset of the pain was gradual and there is reason to believe that other causes, for example, foci of infection elsewhere in the body, were present before the accident occurred, its real cause can frequently be demonstrated. Any possibility of the ailment being of mechanical origin should necessarily be excluded. A man *suffering from flat-feet works under* a mechanical strain and frequently his latent disability manifests itself in the *form of* secondary *lumbar strain* As the casual static flaw is often overlooked, the would-be claimant is only too likely to be stigmatized offhand as a pure malingerer, which is obviously unfair.

In making the physical examination the following points should be considered

1. Local swelling, ecchymosis, and, in cases of long standing, tissue induration, trophic and vasomotor changes.

2. Tenderness to pressure at the site of pain

3 Local muscular spasm and rigidity.

4. Aggravation of the pain by active contraction or passive extension of the affected muscles.

5. Impotence, absolute or relative, of the affected muscles.

6. Correlated phenomena; altered facial expression, pupillary dilatation, accelerated pulse-rate, and raised blood-*pressure*

A patient exhibiting these phenomena is certainly not a malingerer, though he may *still* be exaggerating *his pain and* disability. Pain in the back, of traumatic origin, is intensified by certain movements and not by *others, and inconsistencies* may slip out The following ruse is sometimes of value: With the patient sitting, *the examiner places* his hand on the thigh of the side al-

marked neurotic element in many of these cases, even where there is no intention of malingering Sewell[1] reported a case of hysteria simulating brain tumor. Even in a true traumatic neurosis "lump sum compensation will often clear up a great many of the symptoms."

Injury Simulated by Drugs: Particular diseases are sometimes simulated by the use of drugs Smallpox, for example, may be simulated by the application of croton oil to the surface of the body.

Attempts to produce the effect of jaundice are sometimes made by taking large internal doses of picric acid, this may be detected by the yellow pigmentation which usually appears on the skin *in patches* instead of the generalized coloration of true jaundice, also the urine and stool will be stained yellow and contain picric acid

Alleged General Debility: When general debility is alleged the examiner may have to make a very thorough scrutiny of all the circumstances of the case before declaring that the allegation is a fraud The pitfalls that, in the presence of such allegations, beset a hasty diagnosis of simulation, are tuberculosis, latent pleurisy, diabetes, Bright's disease, cerebral tumor, neurasthenia and ambulatory typhoid. Early tuberculosis for its exclusion needs not only careful clinical examination, but the use of x-rays, bacteriology, and if necessary, sero-diagnosis One of the very best methods of "checking up" a patient, is to weigh him at stated, frequent intervals Latent pleurisy can only be ascertained by leisured and thorough examination Bright's disease and diabetes can be readily detected by urinalysis, but if this were omitted could be easily overlooked. Without ophthalmoscopic examination, the existence of cerebral tumor may easily pass without recognition. To estimate the reality of neurasthenia, a disorder subject to fluctuations, Chavigny lays stress on "the disorders of nutrition, as evidenced by digestive and urinary derangements, periodic loss of hair, and the presence of unusual furrows, as the neurasthenic is usually absorbed in his subjective symptoms and pays little attention to these objective phenomena"

Simulated Good Health

It is often more difficult to detect the existence of disease in a person who presents himself for examination and denies ever having had any, declaring emphatically that he is perfectly well, than to expose one who is healthy and claims to be ill.

In order to pass a physical examination for life insurance or the army, a person may refuse to give a true history or will evade certain questions. An experienced examiner, however, can usually detect a degree of reticence and by skillful cross-examination may elicit a fairly correct history. (See · Industrial Examinations, p. 939, and Life Insurance Examination, p 946)

Careful physical examination and laboratory tests will aid in a diagnosis; but, however skillful an examiner may be, if the applicant wishes to withhold certain information it is easy to overlook conditions such as epilepsy, gastric ulcer, cholecystitis, various gastrointestinal diseases, malaria (between paroxysms) and a host of other conditions which can be accurately diagnosed only with the cooperation of the one examined.

[1] Sewell Trans Western Sect. Amer. Laryngol, Rhinol and Otol Soc, Feb 23, 1922

G. Boschi.[1] The affected limb is grasped and Lasègue's method of extension performed, the angle formed with the plane of the bed when pain is produced, being approximately noted. The performance is then repeated on the healthy limb, and during its movement the pulse is counted, the pupils noted and the cuff of the sphygmomanometer adjusted until the radial pulse is obliterated. The affected limb is now tested a second time, and the patient watched so that he cannot produce pain by other methods, such as biting the tongue. If the extension produces pain, the pupil will undergo a sharp, transitory, but marked dilatation (3 to 5 centimeters). Nervous apprehension may contribute to this dilatation, but if it is more marked when the affected member is extended than when the test is used on the healthy limb, it is strong confirmation of the genuineness of the disability alleged. The pulse may give further confirmation, so if possible, the radial pulse should be felt by an assistant in the arm upon which the sphygmomanometer cuff has been placed, for if genuine pain is experienced, the resulting increase in arterial pressure will cause the pulse to reappear. Care must be taken to prevent the subject from straining with a closed glottis, which would cause a rise in blood-pressure. Normally, an increase of eight or ten beats will occur during pain, and even more if the subject be neurotic. The healthy limb may be used as a control.

Injury to the Special Senses: Malingering in regard to the special senses, sight and hearing in particular, if not so crudely done as to be possible of detection by the tests of ordinary examination, can usually be demonstrated only by specialists in the handling of these particular branches of medicine.

Injury to the Head: *Vertigo is an* important symptom following head injuries, such as fractured skull and concussion of the brain. These patients often complain of severe major symptoms lasting for a long period, namely, vertigo and instability, headaches, weakness, deafness, nervousness and a great many minor troubles. Unless these patients are found to have definite evidence of an organic neurologic condition, they are placed in a class of functional disorders, namely, traumatic neurosis. Yet quite a number of these cases which are otherwise neurologically negative, show very definite evidence of organic disturbance along the vestibular pathways. The neurologic examination, therefore, should not be considered complete without a thorough neuro-otologic examination. If the examination shows definite evidence of disturbance along the vestibular pathways, these findings may be of the greatest help to the neurologist in differentiating between the functional and the organic cases.[1]

When making a neurologic examination in these cases it is well to bear in mind the following facts: (1) Careful technic must be employed and any abnormal responses must be checked. (2) All the findings in the general examination must be correlated in the interpretation of the diagnosis, not permitting the otologist to go too far in his interpretation.[2] (3) There is a

[1] Boschi: Il Policlinico, March 26, 1916.

[1] Fletcher, H. A.: Determination of Disability ... Aug. 23, 1919.

ination. By the former method, the patient is examined successively by the various specialists; the findings of these specialists are then correlated by the internist who reviews the case and advises the examinee as to the findings and also as to what measures should be instituted for the maintenance of health and efficiency or what defects should be corrected in order to regain perfect health or at least minimize the effects of the damage done and check its further progress For several reasons, this method of examination is not always satisfactory The patient has to meet a number of strangers who may be either too unsympathetic or too solicitous, and therefore, he will not, as a rule, be in perfect co-operation with each of the examiners; also when a number of physicians examine the same patient, each examiner holds himself responsible only for so much of the body as comprises his specialty, and because it is often difficult to draw sharp lines of demarcation between the ending of one specialty and the beginning of another, much may be overlooked.

The other and better plan for periodic health examination is to have either the family physician or some other competent physician do the entire examination, and if any special defect is noted requiring special study, the examiner should then direct the examinee as to what to do and where to go This method is advantageous because the patient is apt to be more at ease and will therefore cooperate with the examiner, the examiner will maintain a greater personal interest and therefore be of greater service; also there is less likelihood of major defects being overlooked. Moreover, the patient's mental attitude, when no special mental examination is indicated, can be studied much better by one man than by a group.

In order to examine the patient completely and systematically and to minimize the possibility of omission, the examiner should have a special chart which he may follow when doing general health examinations.

The method pursued in performing a periodic health examination does not differ from a routine, careful medical examination for any other reason. A complete and careful history is important, then a general observation which is followed by a detailed examination of the eyes, ears, nose, throat, teeth and tongue The neck is examined for enlarged glands and pulsations, the chest for expansion, the breasts should be examined for masses, and the lungs are examined in the usual way If there the findings are suspicious of pathology, an x-ray examination should be made The heart is examined for size, rate, type of sounds, and its response to exercise. The blood-pressure is to be taken with a sphygmomonometer If the heart examination indicates an abnormal change an electrocardiogram should be taken The abdomen and its viscera are examined for size and position In the presence of pain or distention, accompanied by prolonged digestive disturbances, the gastrointestinal tract and the gallbladder should be examined by x-ray and the contents of the stomach, gallbladder, and also the feces should be examined by laboratory means Examination of the genitalia, rectum and the peripheral vascular system should be part of every examination

In other words, when some abnormality in any part of the body is detected, no matter how trivial it may

The special applications of physical examination might be indefinitely extended, but enough has been said to suggest to the average practitioner some of the points which need to be covered with especial thoroughness in certain lines of work, and the best methods of examination when the possibility of fraud arises. The actual procedure is the same for all classes of cases, and the need for accuracy, thoroughness and patience is ever apparent.

Periodic Health Examinations

"There is nothing new under the sun" is a saying attributed to the wise King Solomon of Biblical times. Frequently, scientists may not subscribe to that dictum and point out the many recent discoveries, such as bacteria, certain rivers, continents, various planets, metals, radium, gases, etc, but we must remember that all these and many more have always been in existence. They were unknown to us and the fault for not knowing was our own. Periodic health examinations, though a supposedly new form of examination, no doubt existed in earlier civilizations To keep a person well in order to obviate the necessity of restoring him to health after illness is not a new idea As an example of this, it may be observed that in some portions of China, they have had and still have, the very admirable custom of paying the doctor when in good health, and when sick, payment stops This of necessity should induce the physician to "check up" his patients frequently as to their health, manner of living, playing and working. If any defect is noted, while still in the incipient stage, an attempt is made to correct it Likewise, a modification of the habits may be brought about so as to minimize strain. In modern times, in order to preserve health and maintain efficiency, periodic health examinations are being advocated.

A periodic health examination may be defined as a complete physical and mental examination including routine laboratory examinations given at definite intervals to persons irrespective of their state of health In addition to this routine, such special examinations and laboratory tests as may be indicated by the general examination should be performed. This type of examination is used in order to discover abnormalities in their incipiency even before the patient is conscious of any symptoms and is primarily intended for the person in good health, as it is assumed that the sick individual is already under the care of a physician However, not infrequently, it is found that persons suffering from chronic ailments who are well aware of the fact, will continue at their occupations without medical care or attention until they are completely broken down

The benefits derived from periodic health examinations depend upon two factors: (1) The thoroughness of the examination and the examiner's ability to evaluate properly the various facts obtained in the history, physical and laboratory examination; and (2) the thoroughness with which the examinee carries out the advice received.

In various clinics, both private and public, periodic health examinations are conducted by one of two general methods, either by a group of physicians, each a specialist in a particular phase of the examination, or by only one physician who makes the complete exam-

SECTION 15

Laboratory Procedure

appear at first sight, it should be carefully studied with all the aids at our command. The examiner should not hesitate, when necessary, to have the examinee return within several days or weeks for a recheck.

The examiner should keep a comprehensive record, preferably on a record form, of all the findings at each examination so that the findings of successive examinations can be compared with those made at an earlier date.

CHAPTER XXXIII

Urinalysis

The Rôle of Laboratory Examinations in Diagnosis

No matter how thorough a physical examination may be, it does not always suffice to establish a definite diagnosis, as greatly divergent conditions often present similar physical signs In order to assist in differentiating such conditions and to aid in establishing or confirming a positive diagnosis, various laboratory methods should also be employed For example, by physical examination alone it is difficult to determine whether a pleural, pericardial or peritoneal effusion consists of serum, blood or pus. Again, in the presence of cerebrospinal disease, laboratory aid is necessary to determine the character of *the spinal fluid* The *condition of the* urine, the blood, gastric contents, sputum, feces and other excretions and secretions often have to be carefully investigated to aid in proving or disproving a tentative diagnosis The clinical thermometer, the x-rays, the sphygmomanometer, the electrocardiograph, the polygraph, the microscope, the trocar and cannula, the exploratory needle and many other clinical appliances are adjuvants in obtaining the required data

It is not within the scope of this book to set forth the various and intricate methods used in the examination of the bodily secretions and excretions These methods are standardized and their technic can be found in any book on laboratory examination Only the least *complicated tests, those that can be performed* by the average physician and do not require special training in this field of medicine, will be described here. The significance of abnormal laboratory findings in various diseases will, however, be stressed, inasmuch as it is the physician's duty to interpret such findings when they are reported to him from the laboratory.

Method for Collecting and Examining the Urine

For accurate urinalysis, a 24-hour specimen should be obtained. The results from the examination of a single specimen, while valuable, are not conclusive A "night and morning" specimen is preferable to a single specimen, though the 24-hour specimen is the most valuable. The author gives the following instructions to his patients, when a specimen of urine is required:

Single Specimen: The *urine may be* passed in a clean receptacle in the physician's office for immediate examination, or it may be passed elsewhere and collected in a perfectly clean vessel and *four ounces promptly sent to the laboratory.* Wide-mouthed four-ounce bottles especially adapted for this purpose can be obtained at a drug store, and when possible the urine should be passed *directly into the bottle.*

Night and Morning Specimen: (1) The evening specimen is to be obtained in the following manner: Empty the bladder immediately before the evening *meal and discard this urine.* From the urine first passed after the evening meal,

take four ounces and note the hour when voided.

2 The second specimen is obtained from the urine first passed *upon arising* in the morning. Note the hour when the urine was passed.

To Obtain the Total Quantity of Urine Passed in 24 Hours: The day on which the observation is begun, at a definite hour in the morning, the bladder should be emptied, and this urine discarded. *All* the urine passed afterwards is to be collected in a suitable, clean dustproof receptacle and kept in a *cool* place and, preferably, toluene or chloroform added as a preservative. The following day at the same hour, when the bladder was first emptied and the urine discarded, the bladder is again emptied. This urine should be added to complete the total amount for 24 hours, which should be expressed in ounces or cc. After the total 24-hour quantity of *urine has been collected and thoroughly* mixed, four ounces of the mixture should be sent for examination. A label, on which is written the patient's name, address, date, and the time when the urine was passed, should be pasted on the bottle.

Example: Observation begun on January 1st, at 8 A. M. The bladder is emptied at 8 A. M, this urine is discarded; the urine passed during the day and night are saved. The next morning, January 2d, at 8 A M. the bladder is again emptied, and this urine is added to complete the total quantity for 24 hours.

Procedure in Urinalysis: A urinalysis is an important procedure during the course of a patient's general examination. It mav be brief and consist first *only of four steps:*

I. Determination of specific gravity and reaction.

II. Determination of the presence of *albumin*

III. Determination of the presence of glucose.

IV Microscopic examination of a drop of urine so as to note the presence of cells, casts and crystals.

If an entirely negative result is obtained it can then be assumed that the kidneys are functioning sufficiently well If the specimen of urine shows some *abnormality in either one, two or all the* tests performed, then a minute chemical and microscopic examination should be undertaken in a well-equipped laboratory.

Characteristics of Normal Urine

I. *Frequency of urination* in the normal individual depends upon habit and the quantity of urine present, usually *it is about four or five times in 24 hours*

II. *Quantity in 24 hours* is about 1500 cc. or 48 ounces, or approximately 65 cc or 2 ounces per hour

III. *Color* varies from light yellow to dark amber.

IV. *Odor:* Fresh urine is characteristically aromatic; old urine ammoniacal Certain foods import characteristic odors to the urine.

V. *Reaction* is slightly acid (*p*H6)

VI. *Albumin* is not found by the usual laboratory examination

VII. *Glucose* is not found by the usual laboratory examination.

VIII. *Iodine* 25 to 75 µg. (micrograms) in 24 hours.

IX. *Specific gravity* is *1.016 to 1.024*, taken with any standard urinometer.

X. *A slight sediment* of calcium oxalates, phosphates, etc, may be present

Approximate Amounts of Protein, Casts, and Cellular Structures Found in the Urinary Sediments of Normal Men

"THE ADDIS COUNT"

	Range	Average per 12 Hours
Red corpuscles .	0–425,000	65,750
Casts	0–4,700	1,040
Leukocytes and epithelial cells	32,400–1,000,000	322,550
Protein	10–30 mg	

TECHNIC FOR OBTAINING AN ADDIS COUNT

1. Liquids are restricted for 24 hours
2. The 12-hour night specimen is collected in a vessel containing tricresol as a preservative The quantity of urine passed is measured and mixed.
3. Ten cc of the urine is placed in a graduated centrifuge tube and centrifugated for about eight to ten minutes at 1500 revolutions per minute.
4. The sediment is resuspended in a measured quantity of normal saline and a drop is placed on a hemocytometer and the number of formed elements are counted and computed according to a definite formula.

The amount of the increase in the number of formed elements above the normal indicates the extent of the inflammatory process in the kidneys. It differs from the kidney function tests in that while the former determines the extent of the inflammatory process, the latter indicates the amount of kidney tissue that has stopped functioning because of the inflammation

The most abundant constituents of the urine are water, urea and sodium chloride. The acids and bases above mentioned are combined in the urine to form salts, urates, chlorides, sulfates, phosphates, etc

Characteristics of Pathologic Urine

Pathologically, urination may be increased or decreased in frequency; it may contain a greater or lesser amount of all solids excreted or of any one or more of these constituents; or it may contain substances that are found only in abnormal states, *e. g.,* albumin, glucose, various salts, etc.

APPROXIMATE AMOUNT EXCRETED IN 24 HOURS BY A HEALTHY MALE ADULT
(*Hauk and Bergeim*)[1]
(Total Volume in 24 Hours, 1500 cc.)

Constituents	Absolute Weight	Approximate Percentage
Water	1440	96 0
Solids	60 0	4 0
Urea	35 0	2.33
Uric acid	0.75	0 05
Hippuric acid . ..	0.7	0 05
Oxalic acid	0 015	0 001
Aromatic oxyacids	0.06	0.004
Creatinine	1 0	0 07
Thiocyanic acid (as KSCN)	0.15	0 01
Indican	0 01	0 001
Ammonia	0 65	0 04
Sodium chloride	16.5	1 1
Phosphoric acid . . .	2.5	0 15
Total sulfuric acid ..	2 5	0 15
Silicic acid .	0.45	0 03
Potassium (K_2O) . .	2 5	0.15
Sodium (Na_2O)	5 0	0 3
Calcium (CaO) . .	0.25	0 015
Magnesium (MgO) .	0.30	0 02
Iron .	0 005	0 0004

I Frequency of Urination: This may depend upon the quantity of urine passed; the greater the quantity the more frequent is micturition This, however, is not always the case; often but a few drops of urine will be passed at a time, perhaps every one-half hour or even more often, depending upon the state of irritability of the bladder and urethra

Urination is increased in frequency in polyuria of any cause, nervous excite-

[1] Hawk and Bergeim Practical Physiological Chemistry, 9th Edit, P. Blakiston's Son and Co, Philadelphia

ment, disease of the spinal cord, irritation of the bladder (by inflammation, foreign body, *stone, tumor, parasites*), irritation of the urethra or the urinary meatus, enlarged prostate in the male, pregnancy in the female. In children it may occur reflexly because of adenoids, intestinal worms, irritable sphincter and phimosis.

Decreased frequency of urination is seen after profuse sweating, diarrhea, and bleeding; in oliguria or anuria, in uremia; in parenchymatous nephritis; in *brain diseases; in deep coma, and it may* be caused by drug poisoning, *e. g.*, mercuric chloride, oxalic acid, etc.

II. **Quantity:** The quantity of urine passed in 24 hours varies within fairly wide limits; in health usually between 1000 to 1500 cc. or two to three pints (32 to 48 ounces), *i. e*, 1⅓ to 2 ounces for every hour in the 24 In disease it may be increased, diminished or absent for a number of days, depending upon *the condition of the secreting parenchyma* of the kidney and the rapidity of the renal circulation.

Polyuria means increase in the quantity of urine; both the liquid and solid constituents are proportionately increased.

Hydruria is an increase in the watery constituents of the urine, the solids being proportionately *very much diminished.*

Oliguria is a diminution in the total *quantity of urine excreted.*

Anuria means complete suppression of urine.

Polyuria is found after the ingestion of large quantities of fluid (hydruria), and in the following diseases: Diabetes mellitus; chronic interstitial nephritis, *amyloid disease of the kidney*, diabetes insipidus (hydruria), in conditions attended with high blood pressure, in hysteria and often in exophthalmic goiter, and when large exudates or transudates are being absorbed (ascites or anasarca).

Oliguria is noted in acute nephritis in heart disease during the stage of decompensation, in low arterial tension, in cirrhosis of the liver, in the presence of pyrexia and in persistent diarrhea, sweating and hemorrhage.

Anuria may occur either as a result of suspended activity of the kidneys, as in mercuric chloride poisoning and uremia, or because of paralysis of the bladder such as may occur with a spinal *lesion In the latter instances the urine* excreted by the kidneys accumulates in the bladder but is not expelled This condition is easily recognized by palpating the bladder above the symphysis pubis, and is confirmed by catheterization.

III. **Color:** The light or dark straw color of normal urine is due to the *presence of urochrome and urobilin*, substances derived from biliary pigment. Acid urine is usually darker than alkaline (when fresh). In oliguria, as a rule, because of greater concentration, the urine is darker than in polyuria A change in the color of the urine may be the result of certain diseases, the ingestion of various foods or dyes and *particularly of drugs, or of various* metabolic changes.

Pale urine is usually associated with polyuria, and is often seen in cases of diabetes mellitus, diabetes insipidus and chronic interstitial nephritis; also in certain nervous affections, *e. g*, hysteria, epilepsy, nervous *strain; and after* ingestion of large quantities of liquids

Dark urine is usually the result of greater concentration of solids In febrile diseases the dark urine is caused by a

substance known as uroerythrin. It is also seen in cholera and typhus.

Dark green or greenish yellow urine may be caused by the presence of bile (as in obstructive jaundice), or by the ingestion of certain drugs, such as phenol, santonin, salol, guaiacol, resorcin, etc.

Pale urine with high specific gravity is often due to the presence of glucose.

Reddish or orange brown urine may be caused by the presence of blood or bile, or the ingestion of rhubarb, senna, tannic acid, chrysarobin, picric acid, etc

A yellowish tint in the urine may be due to the presence of bile, pus, or some fatty substance; the latter two usually cause a milky appearance

Blood red or pink urine is usually due to the presence of fresh blood Pseudomembranous or chromogenic bacteria may impart a blood-red color to the urine but the absence of red blood corpuscles in an acid urine will differentiate the second condition from the first

Smoky brown urine usually results from ingestion of phenol or the various products of which phenol is a constituent The presence of blood or its derivatives may cause the urine to assume a smoky color

Black urine may be found in melanotic sarcoma, in phenol poisoning, and in alkaptonuria.

White or opalescent urine is due to the presence of pus, chyle, phosphates, fat globules and ammonium urates.

Bluish urine is usually the result of ingesting methylene blue; a bluish colored urine has also been observed in typhoid fever.

Phosphaturia in the presence of hypoacidity will cause the urine to become *turbid when cooling*, and will also produce a *white precipitate on boiling* which disappears by the addition of acetic acid.

Urine which becomes *dark on standing* usually contains resorcin, an end product of phenol ingestion. The presence of alkapton and melanogen will also cause the urine to become *dark or smoky on standing*.

IV. **Odor:** The normal urinary odor of a freshly voided specimen may undergo various changes when exposed for some time to the air. Fresh urine not so exposed may possess an abnormal odor, because of disease or the ingestion of certain foods On standing, the urine develops an ammoniacal odor due to the presence of free ammonia as a result of urea bacterial decomposition.

Fresh Specimens: *Ammoniacal* odor is perceptible in cystitis due to the decomposition of the urine in the bladder. *Putrid odor* results from putrefactive changes in the bladder due to pus or other albuminous substances. *Stale egg or hydrogen sulfide odor* may result from the decomposition of cystine in the urine, which is present in small amount in normal urine and is the principal sulfurized amino acid. *Sweetish or acetone odor* is often found in diabetic urine, starvation and in acidosis *Violet odor* may result from the ingestion of turpentine. Sandalwood oil and copaiba, asparagus, and various other articles of food impart a characteristic odor to the urine.

V **Reaction:** The reaction of a 24-hour specimen of normal urine properly preserved from bacterial decomposition is usually acid, so that *blue* litmus paper immersed into it turns red The hydrogen ion concentration usually varies from *p*H5 5 to 8 0 *p*H6 may be taken as the mean acidity. Sometimes the reaction is neutral or amphoteric—turning

red litmus paper blue and blue litmus paper red. Rarely it is alkaline—turning red litmus paper blue.

The reaction of freshly voided urine depends largely upon the stage of digestion and the kind of food ingested, and also upon the condition of the urinary tract. The acid reaction of normal urine is due to acid salts, chiefly acid-sodium phosphate and not to free acids, because the phosphoric, uric and hippuric acids are combined respectively as phosphates, urates and hippurates. During digestion, the urine is alkaline except in pernicious anemia and other diseases in which achlorhydria is present. As a general rule *gastric hyperacidity produces alkaline* urine, and gastric hypoacidity—as after fasting or because of organic disease—*will produce acid urine.*

The urine of herbivorous animals and vegetarians, whose food has an excess of alkaline salts and organic acids like tartaric, citric, malic, etc., will be rendered alkaline by the oxidization into carbonates of the acid salts. Carnivorous animals and those indulging in much meat or proteins will secret a *highly* acid urine.

Increased acidity of urine may be caused by the following: (*a*) The ingestion of acids (those which are not oxidized to carbonic acid, *e. g.*, the mineral and aromatic acids); (*b*) fevers; (*c*) inflammations of the liver; (*d*) acute articular rheumatism, (*e*) lithemia; (*f*) diabetes; (*g*) uric acid diathesis; (*h*) after violent exercise.

Alkaline urine may be caused by: (*a*) Bacterial decomposition; (*b*) alkaline fermentation of urine in the urinary tract; (*c*) retention of urine in the bladder; (*d*) the constant presence of residual urine in the bladder; (*e*) chlorosis; (*f*) general debility; (*g*) when rapid absorption of exudates or transudates is taking place (the alkaline salts are excreted in the urine); (*h*) the admixture of alkaline secretions, *i. e.*, blood or pus, from the urinary tract with the urine; (*i*) the presence of cystitis or urethritis; (*j*) abnormal condition of gastric digestion; (*k*) ingestion of acid fruits.

If the alkalinity of the urine is due to free ammonia (indicating decomposition) and not to alkaline salts, a strip of red litmus paper when held near the surface of the urine will turn blue without being immersed, or a glass rod dipped in hydrochloric acid and held over the surface of the urine will produce white fumes of ammonium chloride.

VI. **Specific Gravity:** The specific gravity of a normal 24-hour urine usually ranges between 1.016 and 1.024. It indicates the quantity of solids held in suspension Single specimens of normal urine may vary from 1.008 to 1.030 or over, depending upon the quality and quantity of food and water ingested and upon the amount of liquids consumed After copious sweating or severe diarrhea the urine is more concentrated and exhibits a higher specific gravity. In polyuria, because of low concentration, the specific gravity is low, often only 1.005. Polyuria and high specific gravity may indicate glucose or an excess of urea.

Significance of Specific Gravity: *Low specific gravity* may occur in: (*a*) Diabetes insipidus; (*b*) chronic interstitial nephritis; (*c*) cachexia (because of poor metabolism); (*d*) preuremic states (concentration of solids in the blood because of failure of kidney function); (*e*) amyloid disease of the kidney; (*f*) during convalescence from acute nephritis and from acute fevers; (*g*) after ether anesthesia; (*h*) after hysteri-

cal seizures; (i) after excessive drinking of malt and spirituous liquors.

High specific gravity may occur in: (a) Diabetes mellitus (associated with polyuria); (b) excess of urea or sodium chloride; (c) acute nephritis; (d) chronic parenchymatous nephritis; (e) during the crisis of acute fevers, (f) after severe sweating, diarrhea and vomiting; (g) after ingesting rich foods

Methods of Determination: In order to get fairly accurate data of the specific gravity of the urine, a sufficient quantity to fill a urinometer cylinder is obtained. The cylinder containing the urine is placed upon a level shelf or table and a urinometer (hydrometer) is floated in the cylinder The level to which the stem of the urinometer sinks (reading from below upward), is the approximate specific gravity. If a freshly voided specimen is to be examined and the quantity is insufficient to float the urinometer, the urine may be diluted with a known proportion of distilled water, and the specific gravity thus obtained is then calculated so that the specific gravity of the specimen is ascertained

The Method of Estimating Total Solids: Vierordt's Factor The solids excreted in one liter of urine may be approximated in grams by multiplying the last two figures of the specific gravity by 2.2337 grams

Long's Coefficient Multiply the last two figures of the specific gravity of the urine by 2.6 The result will represent the number of grams of solids in 1000 cc. of urine.

Trapp's Formula. The last two figures of the specific gravity are multiplied by 2; the results represent the proportion of solids in one liter of urine *Example* If the specific gravity is 1.022, 22 times 2 equals 44. Hence there are 44 parts of solids per 1000 cc. of urine.

Bird's Formula The last two figures of the specific gravity represents about the number of grains of solids in a fluid-ounce of urine. *Example:* A specific gravity of 1.022 would contain about 22 grains of solids to the ounce of urine

VII. Sediments and Their Significance in the Urine: Urine when allowed to remain in a vessel undisturbed for some time will usually throw down a precipitate. For laboratory examination the urinary sediments are obtained by centrifugating the specimen. The sediment may contain the normal organic and inorganic constituents and pathologic substances, *i. e.,* shreds, epithelial cells, blood corpuscles, bacteria, casts, albumin, etc

A *"brick dust"* sediment in the urine which disappears on heating is usually due to free urates and uric acid.

A *white flocculent* precipitate, not dissolved by heat, but soluble on the addition of dilute acetic acid is due to calcium and magnesium phosphates (basic phosphates).

A *slight deposit* not soluble in dilute acetic acid, heat or ammonia, but soluble in hydrochloric acid when heated, may be due to oxalates (readily confirmed by microscope).

Constituents of the Urine and Their Clinical Significance

Urea: This is the principal end product of protein metabolism It is the most abundant constituent of the *organic solids* excreted by the kidneys. The normal daily excretion for an adult averages from 30 to 35 grams, depending primarily on the quantity of protein in the diet. Thus in an average diet containing 120 grams of protein a day, the urea excretion would be about 30 grams

On a low protein diet of 50 grams per day the urea excretion may be 8 to 10 grams. Denis and Borgstrom in 1924 completed a three-year study in New Orleans, and found that 233 male medical students showed a daily urea excretion of about 20 grams

Increased urea in the urine is seen in: (*a*) Increased protein intake; (*b*) fevers, especially on loss of weight, (*c*) after pregnancy; (*d*) during parturition, (*e*) after drinking large quantities of beer or water.

Decreased urea seen in (*a*) Low protein intake; (*b*) reduced elimination, (*c*) pregnancy; (*d*) convalescence (gain in weight); (*e*) disease of the liver.

In recent years the practical information available for diagnostic purposes from chemical analyses of the blood is supplanting the quantitative determination of some of these constituents in the urine. This subject is considered in detail under the heading of Blood Chemistry (SEE: p. 1007)

Uric Acid: This name is a misnomer because it is not a typical acid; that is, it does not ionize to any extent and is almost completely insoluble in water. Its salts are, however, soluble in water

Increased elimination of uric acid may occur: (*a*) After the ingestion of large quantities of nitrogenous food (liver, kidneys, brain); (*b*) in gout; (*c*) in acute articular rheumatism; (*d*) in leukemia, and (*e*) after exercise.

Decreased elimination is seen in. (*a*) Those living on a vegetable diet; (*b*) in nephritis; (*c*) in lead poisoning, and (*d*) in chlorosis.

Chlorides: Sodium chloride is the most abundant of all the *inorganic constituents* excreted by the kidneys and is second in quantity only to urea. The quantity passed in the urine in 24 hours varies from 10 to 16 grams, or approximately one per cent. The chlorides in the urine are derived from two sources: (1) Principally from the food and (2) a small quantity from the process of catabolism of the tissues.

Increased chlorides in the urine occur: (*a*) As a result of ingestion of sodium and potassium chloride; (*b*) during the absorption of exudates, (*c*) in diabetes insipidus; (*d*) during the stage of convalescing from fevers; (*e*) after the crisis in lobar pneumonia; (*f*) after epileptic seizures; (*g*) in the afebrile stage of intermittent fever, (*h*) after chloroform anesthesia; and (*i*) after drinking large quantities of water.

Decreased chlorides in the urine usually occur: After strenuous exercise and in the presence of nephritis with edema; in febrile diseases; in starvation; in cachexia, in diarrhea; during the formation of exudates and transudates; in nephrosis; in anasarca, and in acute atrophy of the liver.

An increase in the output of chlorides *in the urine during the course* of a febrile disease indicates an improvement. A *diminished output of chlorides in non*-febrile disorders points to a serious condition (Sahli). The value of chloride determinations in the urine is limited. In central pneumonia, where physical signs are lacking or doubtful, a great decrease in the chlorides affords corroborative evidence of some value. The qualitative test usually suffices for this purpose, a known normal urine being used as a control.

Phosphates: From 2 to 3 grams of phosphoric acid in the forms of sodium, calcium and magnesium phosphate are excreted in 24 hours, the greater part coming from the ingested food.

Increased Output of Phosphates in the Urine occurs: (*a*) During convalescence from acute fevers; (*b*) in diabetes mellitus; (*c*) in diabetes insipidus, (*d*) in leukemia; (*e*) in phosphatic diabetes (Anders and Boston); (*f*) in bone disease; and (*g*) after the administration of such drugs as alcohol, chloral, or chloroform, vegetable acids and the bromides; and (*h*) recently it has been shown that in violent exercise, mental strain, anxiety and after hot baths the phosphate parallels the increase in acid excreted.

Decreased excretion of phosphates is principally observed in nephritic acidosis and must be confirmed by determining the phosphorus and CO_2 content of the blood plasma or serum. Any marked and persistent phosphate retention is a bad prognostic sign

Sulfates: The normal 24-hour specimen of urine should contain from 2 to 3 grams (30 to 45 grains) of sulfate combined in two groups· (1) The mineral, inorganic or preformed sulfates occurring as sodium and potassium sulfate, and (2) the organic, conjugate or ethereal sulfates, occurring as phenol potassium sulfate, skatoxyl potassium sulfate and indoxyl potassium sulfate (indican) In a 24-hour specimen the amount of inorganic sulfates is to the organic as 10 to 1 The quantity of sulfates in the urine is influenced to a large extent by the amount of protein food ingested and by the extent of tissue destruction that is taking place

Increase of sulfates in the urine may occur in those who indulge in too rich a protein diet and also in the following conditions (*a*) Acute febrile disease; (*b*) meningitis, (*c*) acute myelitis; (*d*) progressive muscular atrophy; (*e*) diabetes mellitus, (*f*) diabetes insipidus; (*g*) eczema; (*h*) myeloid leukemia; (*i*) in wasting diseases such as carcinoma of the esophagus. (*j*) The ingestion of drugs such as salicylates, bromides, the coal-tar products and morphine, also have a tendency to increase the phosphates in the urine. Anders and Boston point out a feature of clinical importance: Namely, whenever the percentage of hydrochloric acid is lessened in the stomach, the ethereal sulfates are increased in the urine, consequently an increase is present in gastric fermentation.

Decreased sulfates in the urine occur in those who exist largely on a vegetable diet The condition is also seen after diarrhea, in depleting conditions and when the gastric juice is found to contain an excess of lactic and butyric acid The sulfate excretion is always decreased in the slowing up of metabolic activity

Sulfur: Loosely combined sulfur in the urine is found in bone disease (myelomata), with associated albumosuria

Indican (Indoxyl potassium sulfate): In normal urine this substance occurs only as a trace, 4 to 20 mg. in 24 hours A high meat diet causes an increase and a carbohydrate diet a decrease An excess of indican in the urine (*indicanuria*) occurs· (*a*) As a result of intestinal putrefaction; (*b*) in carcinoma of the stomach, or other diseases of the stomach associated with an absence of hydrochloric acid; (*c*) in peritonitis; (*d*) chronic and acute obstruction of the bowels or any condition that slows or stops intestinal peristalsis; (*e*) acute infectious disease; (*f*) pulmonary gangrene; (*g*) gangrene of the extremities; (*h*) emphysema; (*i*) puerperal sepsis; (*j*) typhoid fever; (*k*) ob-

On a low protein diet of 50 grams per day the urea excretion may be 8 to 10 grams. Denis and Borgstrom in 1924 completed a three-year study in New Orleans, and found that 233 male medical students showed a daily urea excretion of about 20 grams.

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Phosphates: From 2 to 3 grams of *phosphoric acid in the forms* of sodium, calcium and magnesium phosphate are excreted in 24 hours, the greater part coming from the ingested food.

cause the presence of albumin in the urine in quantities sufficient to be detected by the usual clinical laboratory methods generally indicates disease of the kidneys. The significance of albuminuria in kidney conditions depends upon the quantity of albumin and other urinary findings, *e. g.*, specific gravity, quantity in 24 hours, casts, blood, etc. The patient's history and the data obtained by physical examination and chemical analysis of the blood are also to be taken into consideration when the significance of albuminuria is to be determined.

Albumin in the urine, as has just been mentioned, may occur as a result of increased permeability of the renal epithelium of both the glomeruli and tubules permitting the blood proteins to pass into the urine, or because of disease of the renal epithelium which not only permits greater permeability but also causes a certain amount of inflammation or degeneration of the kidney substance.

Albuminuria is found in the various kidney lesions, in certain diseases of the blood, in cardiac decompensation, in fevers, in toxemias and in poisoning by certain drugs, in local inflammations of the genitourinary tract and at times in apparently healthy individuals

Functional or Transient Albuminuria: This is a term applied to a condition in which the occasional finding of albuminuria is the only symptom; the person is apparently healthy and is feeling well, and on careful examination does not present any evidence of pathology. It seems hardly believable that a perfectly normal kidney should manifest abnormal permeability particularly so when one realizes that kidney function may be reduced to at least 50 per cent without showing clinical evidence of disturbed function. This is often noted when one kidney is removed; the remaining kidney, if well, carries on normal function However, transient albuminuria does exist and it is found frequently during the period of puberty or adolescence, particularly in weak and anemic children. In apparently healthy adults albuminuria may be found after exercise, after cold baths and during digestion; also, on change of posture from the recumbent to the erect and is usually manifested on arising in the morning Spinal curvature, especially lordosis, also has a tendency to cause albuminuria

The diagnosis of transient albuminuria is based upon the occasional presence of albuminuria, the urine in all other respects being normal and the patient presenting no other abnormality.

The *albuminuria of fatigue,* which occurs intermittently and is slight in amount, appears only after prolonged, fatiguing exercise, such as hiking, running, horseback riding, etc, and generally disappears with rest This may be associated with casts.

The *digestive albuminurias* are those which arise or become accentuated during the process of digestion, whether the subjects be dyspeptic, enteritic, or apparently normal. The relationship of cause to effect can be established only by repeated fractional analysis of gastric juice withdrawn at various stages of digestion, every precaution being taken to eliminate orthostatic albuminuria.

The *cyclic albuminurias* are those appearing in a cyclic manner, at certain periods of the day, generally between 1 and 3 P. M. According to Teissier and Pavy, they seem to be dependent upon

structive jaundice; (*l*) intestinal parasites (Diphyllobothrium latum), and (*m*) in oxaluria.

Oxalates: The daily normal quantity excreted in the urine is about 15 to 20 *mg* Because of its insolubility (one part of calcium oxalate requires 500,000 parts of water), a deposit of oxalate crystals in the urine on standing does not always indicate oxaluria. Such a deposit may be due to the ingestion of certain vegetables and fruits, *e. g*, cabbage, *carrots, spinach, tomatoes, string beans, onions, celery, asparagus, rhubarb,* apples and grapes The imperfect oxidation of carbohydrates will cause an increase in the excretion of oxalic acid Increased oxalates in the urine, when not caused by the food ingested, may be due to an oxaluric diathesis, dyspepsia, debility, gout, lithemia, so-called neurasthenia, chronic skin diseases, constipation and may occur in the extremes of life (children and aged) and in hemophilia. Gormandizing and lack of exercise are two very important factors in the production of oxaluria

Creatinine: This is a normal constituent of urine, averaging from 1 to 1.5 grams in 24 hours, the exact amount depending upon the food intake and, in the opinion of Shafer, also on the muscular metabolism

The creatinine content of urine is said to be increased in typhoid fever, typhus, tetanus and pneumonia and decreased in anemia, chlorosis, paralysis, muscular atrophy and in advanced degeneration of the kidneys.

Creatine: A small amount of this substance may be found in normal adult urine It is increased in normal children, and in malnutrition, exophthalmic goiter, Addison's disease, and pregnancy. It is decreased in hypothyroidism. The normal ratio between creatine and creatinine is 1 : 10. In hypothyroidism it is 1:8 or 1:5. In hyperthyroidism it is 1·15, or 1:20.

Hippuric Acid: This is possibly formed by the liver from glycine and benzoic acid and is excreted by the kidneys The average quantity eliminated in 24 hours is from 0 7 to 1 0 gram (10 to 15 grams). This amount may be increased by a vegetable diet particularly rich in benzoic acid (prunes, cranberries, *bilberries,* greengages). The ingestion of benzoic acid markedly increases the output of hippuric acid It is decreased in certain nephropathies and particularly in certain liver diseases (SEE Liver Function Tests, p. 1040).

Cystine: A trace of this substance is found in normal urine It is increased *in phosphorus poisoning* and acute yellow atrophy of the liver Chronic systinuria may be a congenital anomaly of metabolism. There are instances recorded where several members of the same family have been thus affected. Cystinuria is due to the inability of the body to catabolize sulfurized amino acids to sulfates and neutral sulfur.

Albumin and Tests for Albuminuria

Albuminuria may be *renal, or extrarenal* (accidental).

Renal albuminuria occurs as a result of some changes in the epithelial cells of the kidneys which render them abnormally pervious to the proteins of the blood *Accidental or extrarenal albuminuria* is caused by contamination of normal urine with pus, blood or chyle Renal albuminuria is usually associated with tube casts and is found in all forms of nephritis.

Albuminuria is a sign which should never be allowed to pass unnoticed, be-

the albuminuria was apparently due to a temporary or functional derangement, therefore a toxic nephritis, and if on the other hand the kidney symptoms remain after the patient has apparently recovered from the primary disease it is taken as evidence of *true nephritis*

Albuminuria in Nephritis: In the various nephridites, albuminuria is a prominent symptom. The quantity of albumin varies with the type of kidney lesion, a diagnosis of a definite type of nephritis, however, cannot be made by considering only the quantity of albumin present in the urine Other urinary findings, kidney function tests, blood chemistry data and a physical examination of the patient are necessary for the determination of the precise kidney lesion.

Acute Diffuse Nephritis: In this type of kidney lesion, the 24-hour output of urine is greatly diminished, ranging from 100 to 500 cc The urine is dark in color and often contains blood The specific gravity is high, *albumin occurs in large amounts* and all types of casts (*i e*, hyaline, granular and bloody) are present in great abundance The blood chemistry reveals retention of urea-nitrogen, nonprotein nitrogen, creatinin, uric acid and chlorides

The patient generally runs a febrile course, is very edematous and usually anemic

Chronic Nephritis: Two main groups of chronic nephritis are to be considered from the standpoint of urinary findings particularly of albumin

1 *Chronic parenchymatous* or *chronic tubular nephritis* or *chronic nephritis with edema and salt retention* In this type of nephritis the quantity of urine *excreted in 24 hours is scanty*; the specific gravity is high Albumin is present in large quantities as are also all varieties of tube casts. The blood chemistry reveals retention of chlorides and, as a rule, no nitrogen retention unless the condition is a diffuse nephritis when evidence of retention of nitrogenous products may be found.

2. *Chronic interstitial* or *chronic glomerular nephritis*, or *chronic nephritis with hypertension and nitrogen retention* and *without edema or salt retention*

In this type of nephritis the quantity of urine passed in 24 hours is large; the urine is light in color, of low fixed specific gravity and contains but a *trace of albumin* and only a few hyaline and granular casts. The blood chemistry reveals retention of uric acid, urea nitrogen, nonprotein nitrogen, creatinin and *no salt retention* The patient is, as a rule, not edematous, the blood pressure is high, and there is a tendency toward uremia.

Albuminuria of Passive Congestion: *Passive congestion* of the kidneys secondary to cardiac decompensation will usually present a fairly large quantity of high colored urine, of high specific gravity, containing a large amount of albumin and many casts of all types On physical examination it will be found that the patient is essentially a cardiac sufferer and that the albuminuria is probably secondary to disease of the cardiovascular system It is, however, often difficult to differentiate definitely between cardiac decompensation *per se* and cardiorenal vascular disease.

Albuminuria of Nephrosis (Epstein): Nephrosis, when uncomplicated by nephritis, usually presents a very pale and very much edematous young person with hypotension and low basal metabolism, whose excretion of urine is scanty and of moderately high specific

some degree of insufficiency (or debility), of the liver and kidneys.

In *orthostatic albuminuria*, the standing posture is the sole necessary and sufficient factor of the albuminuria, which passes off when the subject reclines. It is especially frequent in childhood.

The *intermittent and minimal type of albuminuria*, well described by its name, is a slight (0.1 to 0.2) and intermittent albuminuria, which appears and disappears without any sort of periodicity, independent of all fatigue, digestive process, or body posture; this constitutes, according to Sajous, the most cryptogenic of all the forms of albuminuria

Malingerers may mix normal urine with egg white or other albuminous substances in order to claim albuminuria, or they may inject albuminous substance per urethra into the bladder. When malingering is suspeceted, several specimens of urine are to be examined at various times. In the presence of normal blood chemistry and in the absence of tube casts or of blood or pus, albuminuria may be disregarded.

Toxic Albuminuria: This is a condition in which the renal epithelium is disturbed either (1) by a toxic substance produced within the body, or (2) by a poison introduced into the body from an outside source.

1. *Toxic substances originating in the body* may cause mild or severe kidney disturbance depending upon the type of toxin, the quantity and the length of time the toxin has been in operation.

Albuminuria of pregnancy is an example of toxic albuminuria; care must be taken to differentiate a true albuminuria of pregnancy from a preëxisting nephritis or pyelitis The history of normal urine, normal blood pressures and the absence of edema before pregnancy and the gradual oncoming of these symptoms with increasing severity as pregnancy advances is of diagnostic importance. A study of the other urinary findings, such as pus, casts and blood in the urine and the determination of kidney function as well as a study of the blood chemistry are of both prognostic and diagnostic value

Diabetes, chronic constipation, acute and chronic inflammations and suppurations, acute febrile diseases and many chronic diseases may during their course present albuminuria The severity of the albuminuria is necessarily dependent upon the amount of toxemia produced and its action upon the kidneys In all forms of toxic albuminuria, irrespective of their severity, the albuminuria will disappear when the underlying cause is removed, providing no permanent damage was done to the kidney structures

2. *The ingestion of poisons* either by mouth, hypodermically, absorption through the skin or by inhalation may cause a temporary strain upon the kidneys with the resultant albuminuria. If no permanent kidney damage is effected. the albuminuria will disappear when the toxic substances are eliminated from the system. During the time that the toxins are operative, it is often impossible to differentiate between a true nephritis and a toxic nephritis, because in severe cases of both varieties there may be urinary retention, large quantities of albumin, many casts of all types, and the blood may reveal retention of nitrogenous products. The final diagnosis in such cases can only be made after the disease has run its full course, thus a "post hoc propter hoc" reasoning is adopted. If the kidney symptoms are cleared up on the recovery of the patient,

the albuminuria was apparently due to a temporary or functional derangement, therefore a toxic nephritis, and if on the other hand the kidney symptoms remain after the patient has apparently recovered from the primary disease it is taken as evidence of true nephritis

Albuminuria in Nephritis: *In the* various nephridites, albuminuria is a prominent symptom The quantity of albumin varies with the type of kidney lesion, a diagnosis of a definite type of nephritis, however, cannot be made by considering only the quantity of albumin present in the urine Other urinary findings, kidney function tests, blood chemistry data and a physical examination of the patient are necessary for the determination of the precise kidney lesion

Acute Diffuse Nephritis: In this *type of kidney lesion, the 24-hour output* of urine is greatly diminished, ranging from 100 to 500 cc The urine is dark in color and often contains blood The specific gravity is high, *albumin occurs in large amounts* and all types of casts (i e, hyaline, granular and bloody) are present in great abundance The blood chemistry reveals retention of urea-nitrogen, nonprotein nitrogen, creatinin, uric acid and chlorides

The patient generally runs a febrile course, is very edematous and usually anemic

Chronic Nephritis: Two main groups of chronic nephritis are to be considered from the standpoint of urinary findings, particularly of albumin

1 *Chronic parenchymatous* or *chronic tubular nephritis* or *chronic nephritis with edema and salt retention* In this type of nephritis the quantity of urine excreted in 24 hours is scanty; the specific gravity is high Albumin is present in large quantities as are also all varieties of tube casts. The blood chemistry reveals retention of chlorides and, as a rule, no nitrogen retention unless the condition is a diffuse nephritis when evidence of retention of nitrogenous products may be found.

2. *Chronic interstitial or chronic glomerular nephritis,* or *chronic nephritis with hypertension and nitrogen retention* and *without edema or salt retention*

In this type of nephritis the quantity of urine passed in 24 hours is large; the urine is light in color, of low fixed specific gravity and contains but a *trace of albumin* and only a few hyaline and granular casts. The blood chemistry reveals retention of uric acid, urea nitrogen, nonprotein nitrogen, creatinin and no salt retention. The patient is, as a rule, not edematous, the blood pressure *is high, and there is a tendency toward* uremia.

Albuminuria of Passive Congestion: *Passive congestion* of the kidneys secondary to cardiac decompensation will usually present a fairly large quantity of high colored urine, of high specific gravity, containing a large amount of albumin and many casts of all types On physical examination it will be found that the patient is essentially a cardiac sufferer and that the albuminuria is probably secondary to disease of the cardiovascular system. It is, however, often difficult to differentiate *definitely* between cardiac decompensation *per se* and cardiorenal vascular disease.

Albuminuria of Nephrosis (Epstein) · Nephrosis, when uncomplicated by nephritis, usually presents a very pale and very much edematous young person with hypotension and low basal metabolism, whose excretion of urine is scanty and of moderately high specific

gravity, containing an abundance of albumin and globulin, the *latter being* nearly twice as great in quantity as the albumin Casts are usually present in large numbers, but only of the hyaline and granular varieties; and blood casts are conspicuous by their absence In the *early stages of this affection, the blood* chemistry reveals chloride and cholesterol retention and practically normal nitrogenous end product values

Albuminuria is also found in local inflammation or injury to the kidney substance, the ureter, the bladder or the *urethra The presence of blood in the* urine as a result of injury anywhere along the genitourinary tract or contamination during menstruation will give a positive albumin reaction. The nature of the albuminous substance may be *determined by tests for hematuria*

Tests to Detect Albumin in the Urine: Minute quantities of albumin are probably present in normal urine, since urine always contains a variable number of cellular elements derived from *the urinary tract.* Occasionally a specimen of urine containing such a slight trace of albumin as to escape detection may show a number of casts. It is, therefore, unsafe to depend only upon chemical *examination The quantity of albu*min present in normal urine is so minute that it requires a most delicate test to show its presence. The usual clinical tests for albuminuria fail to detect these minute quantities but are nevertheless *sufficiently* accurate to determine albumin for clinical purposes. Of these tests two are most important, and also the simplest to perform: (*a*) Heat and acetic acid test, and (*b*) *cold nitric acid test or its modifications.* When in doubt as to their accuracy, the more delicate tests may be employed.

(a) Heat and Acetic Acid Test: Fill an ordinary clean test tube (preferably pyrex) *two-thirds full of urine,* heat the upper part of the test tube on a slow flame (hold the tube by the lower end) to boiling. If a white precipitate forms in the boiling urine, add 5 or 6 drops of 3 per cent acetic acid solution. *If the white precipitate persists or becomes* more dense it indicates albumin and if the precipitate disappears on the addition of dilute acetic acid, it indicates calcium phosphate or carbonate. Copaiba, turpentine, benzoin, etc., may on *boiling cause a cloud which is readily* dissolved in alcohol.

(b) Cold Nitric Acid or Roberts' Solution Test (Roberts' solution consists of nitric acid, one part; saturated solution of magnesium sulfate, nine *parts*)*: Pour a small quantity of nitric* acid or Roberts' solution in a test tube and allow some urine to flow slowly down along the inner side of a test tube so that it forms a layer above the acid *If at the point of contact between the* acid and urine a white ring is formed, it is indicative of albumin

Boston's modification simplifies this procedure About one inch of urine is drawn up in a clear pipet. *The upper end of the pipet is closed* with the index finger to prevent the urine from spilling. It is then inserted in a bottle containing nitric acid or Roberts' solution When the acid has reached above *the level of the urine, the finger is removed* so that the acid enters the tube The index finger is again applied to the upper end of the pipet and it is thus withdrawn from the bottle In the presence of *albumin, a white ring is visible* at the point of contact between the urine and nitric acid

Fallacies to be Avoided: (1) *Resin.* If the patient is taking copaiba or similar

drugs, enough of the resin may be excreted in the urine to form a diffuse white cloud above the nitric acid. Therefore the nitric acid test should be checked up by the heat test in all cases of suspected albuminuria.

(2) *Albumoses*· These generally occur in association with albumin; should they occur alone the ring formed at the junction of the urine and nitric acid will disappear with warming, to reappear on cooling; and there will be no cloud with the heat test.

(3) *Bence-Jones' Albumose* This occurs without albumin in cases of multiple myelomata and gives a white ring with nitric acid that disappears on warming, to reappear on cooling, with the heat test a dense cloud appears when the urine is heated to about 60° C. and disappears on further heating to the boiling point.

(4) *Nucleoalbumin* The ring formed by the nitric acid test is not in contact with the nitric acid but is higher up, and diffuse; there may be real difficulty in differentiating it from albumin, because both are precipitated by acetic acid, and may therefore give a haze with the boiling test (SEE: p. 980, "Boston's Modification")

(5) *Urates* These may form a cloud when in contact with nitric acid if the urine is very concentrated, the cloud will disappear on gentle warming, and reappear on cooling, so that it may also be mistaken for albumose, this mistake may be avoided by diluting the urine with plain water before the nitric acid test is employed

(6) *Urea Nitrate*· If the urine contains a large percentage of urea, a crystalline deposit of urea nitrate may form at the nitric acid-urine junction, as a rule, the crystalline nature of the ring is obvious on inspection; but in case of doubt the urine should be diluted and the test repeated.

It does not matter which one of the tests is most relied upon for the detection of albumin when the result is negative; but before a positive deduction that a specimen of urine contains albumin is drawn, both the boiling and acetic acid, and the cold nitric acid test should be positive.

Glycosuria (Sugar in the Urine) and Tests for Glycosuria

Reducing sugars may be found in quantities up to 0 2 per cent in urines of perfectly normal individuals and even up to 0 3 per cent in concentrated urines (sp. gr. 1.025 or above); therefore when glycosuria is demonstrated qualitatively, a quantitative test should be made, in order to determine the exact amount present Also a blood sugar test and occasionally a glucose tolerance test should be done so as to determine whether the glycosuria is the result of hyperglycemia or due only to a lowered kidney threshold for glucose

Glycosuria is a prominent sign in diabetes mellitus. When glycosuria is constant on a mixed diet, diabetes mellitus may be inferred, but it should be confirmed by determining the blood sugar concentration or by a sugar tolerance test. In diabetes, glycosuria may be accompanied by polyuria, urine of high specific gravity, hyperglycemia, excessive appetite and thirst, emaciation and weakness

Transient glycosuria may occur in the obese, and in individuals undergoing prolonged mental stress as is evidenced by the frequency of these findings in students at examination time.

Alimentary glycosuria may follow the consumption of large quantities of sugars and starches.

Temporary glycosuria is observed during convalescence from acute febrile diseases, such as typhoid fever, influenza, scarlet fever, measles and pneumonia, or *diseases of the meninges, brain and spinal cord.*

Renal glycosuria shows persistently the presence of glucose in the urine and is not accompanied by hyperglycemia The determination of the respiratory quotient and a sugar tolerance test are essential in differentiating this condition from diabetes mellitus

Cerebral Glycosuria: In the presence of certain types of cerebral tumors, in cerebral hemorrhage, in acromegaly, in some of the encephalopathies and in *some types of meningitis, glycosuria may* be a constant finding. Glycosuria may also occur in thyrotoxicosis, adenoma of the adrenals, pancreatic tumors, and *during pregnancy.*

Tests for Sugar: The tests most generally employed to determine the presence of sugar in the urine are Fehling's and Benedict's *tests, the fermentation* test and the Galatest

Benedict's Test: Place 5 cc. of Benedict's solution in a test tube with *eight to ten drops of urine Boil thoroughly* and allow to cool spontaneously. If glucose be present, the entire body of the solution will show a precipitate *ranging from green to red in color, ac*cording to the sugar content of the urine. In the absence of sugar, the solution remains quite clear, or shows only a faint *bluish turbidity.*

Fehling's Test: Fehling's solution is ordinarily readily purchasable.

To about 5 cc. of hot Fehling's solu*tion, add a few drops of urine, heat and* continue adding urine, a few drops at a time, until there are equal quantities of urine and Fehling's solution. The pres*ence of sugar will be indicated by a red* or yellow precipitate. If in doubt, allow the tube to stand, and any sugar present will precipitate to the bottom of the tube

Fermentation Test: *If* the result of *either of the foregoing tests is doubtful,* it should be confirmed by a *fermentation test.* Special fermentation tubes, or ordinary test tubes may be used Mix the *urine to be examined with a sixteenth* of a cake of fresh compressed yeast and place in one tube. Fill a second tube control with normal urine or water *mixed with a like amount of yeast* The two tubes are placed in an incubator or kept at room temperature If glucose be present, gas will form in the upright of *the fermentation tube, this manifestation* being valuable, however, only when no gas forms in the normal urine. If ordinary test tubes are used, the openings *must be immersed in a* beaker of the same urine which each contains, the opening being downward

Galatest: This method is a fairly re*liable, convenient and rapid* method for qualitative determination of sugar in the urine A small quantity of Galatest powder is placed upon a piece of white paper, *and one drop of urine* is dropped onto the powder A positive reaction constitutes an instantaneous change of color from white to gray or black

The greater the concentration of sugar in the urine, the darker is the color reaction, the range being from 0 2 per cent to 1 per cent or more

Caution: The powder is extremely caustic; it is a bismuth compound in an alkaline medium (caustic soda)

Lactosuria: *This is frequently found during pregnancy* and lactation, and

more readily identified by its osazone crystals.

Pentosuria: This may accompany glycosuria; opium habitués frequently show pentosuria. Pentose does not ferment, and forms typical osazone crystals.

Osazone Crystals: These are obtainable when urine containing sugar is heated in the presence of phenylhydrazin and acetic acid

For **Sugar Tolerance Tests** see p 1012.

Acetone and Diacetic Acid

Acetone and diacetic acid when occurring together in abundance in a diabetic person are a danger signal requiring active treatment. Acetone or diacetic acid may be present in minute amounts in the normal 24-hour urine but is increased in carbohydrate starvation. Its presence in larger quantities indicates some metabolic disturbance. However, it must be remembered that in diabetes complicated with impermeable kidneys, acetone must be tested for in the blood. Generally, the strong acetone odor on the breath is unmistakable.

"A differential diagnosis is sometimes necessary between uremic and diabetic coma, as ketosis may occur for some incidental reason in a nephritic patient. Tests for betaoxybutyric acid are scarcely practicable, therefore qualitative reactions for acetone and acetoacetic (diacetic) acid are used. Quantitative determinations of acetone bodies are not needed for practical purposes even in diabetes" (F. M. Allen)

Gerhardt's Test for Acetoacetic Acid: The simplest way of performing this test is to layer a few cubic centimeters of ferric chloride solution (about 10 per cent strength), under a somewhat larger quantity of urine in a test tube. The pale precipitate of phosphates does not hinder the recognition of the true reaction which is a color ring of Burgundy red, ranging from a faint tint to almost black. Some crude idea of the degree of the ketonuria is thus obtained but all attempts at even approximate quantitative calculations are fallacious.

The administration of drugs, especially salicylates, antipyrin and other coal-tar products, will give false reactions. The color given by the drugs is often atypical, but the distinction is best made by boiling the urine a few minutes and repeating the test after cooling. The false reaction remains present: but the true acetoacetic acid is quickly changed into acetone by heat, so that the test after boiling is negative.

Rothera Test: Pour a small quantity of urine in a test tube and add a large excess of ammonium sulfate crystals; a few drops of fresh five per cent sodium nitroprusside solution, and finally a few drops of ammonia water. Through all these steps the tube should be shaken to maintain a full saturation with ammonium sulfate, and some crystals should still remain at the bottom at the end of the process. A positive reaction consists in a permanganate color, ranging from the palest perceptible tint to almost black. It is necessary to wait almost five minutes to make sure that the maximum intensity of color is developed. Quantitative judgment is based upon the quickness with which the color develops as well as its intensity. For economy, when numerous tests are performed it is satisfactory to use only two or three drops of nitroprusside solution with a few drops of urine in a very small test tube and one or two drops of ammonia. A fresh nitroprusside solution means one possessing its original red color, which

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with the blood or only the first and last parts. Bleeding of bladder origin results from cystitis, traumatism to the bladder by a foreign body, such as stone; or because of varicosities, gangrene, papilloma, carcinoma, angioma, or other neoplasms A cystoscopic examination should be made in all cases of hematuria, to determine the cause and origin of the bleeding.

Ureter Origin: The urine and blood are well mixed; occasionally wormlike clots may be found. Bleeding may result from the attempt to pass a stone, after ureter catheterization, and less frequently because of the presence of profuse oxaluria

Kidney Origin: The blood and urine are intimately mixed. The bleeding may originate in both kidneys, as in acute nephritis, or in one kidney only, as in tuberculosis, cancer, hypernephroma or stone in the kidney In the presence of hematuria of kidney or ureter origin, the ureters should be catheterized, so that the urine excreted by each may be separately examined, also pyelographic studies should be made An x-ray examination other than a pyelographic study may be useful in determining the presence of stones and the apparent size of the kidney When a lesion of the urinary apparatus is suspected, and there is no macroscopic blood in the urine, a microscopic examination and the Benzidine test for blood should be made.

Hemoglobinuria: The presence of hemoglobin free from red blood corpuscles in the urine may be due to a variety of conditions. Purpura, scurvy, pyemia; typhus fever, and poisoning by certain drugs, such as arsenic, phosphorus, phenol, mercury, chloral, potassium chlorate, etc Hemoglobinuria in an acid urine indicates that the trouble is outside of the urinary tract.

Pyuria

Pus in the urine may result from a lesion in the urethra, bladder, ureters, kidneys or come from outside sources Butler[1] classifies pyuria as follows:

(*a*) **Urethritis:** If the pyuria is due to urethritis (simple or gonorrheal), a drop of pus may be squeezed from the urethra, and if the two-glass test is employed, the first portion of urine voided contains pus, while the second is clear. If the second portion also contains pus in even larger quantity and is alkaline, the presence of a coexisting cystitis may be inferred, A gonorrheal ulcer, or a suburethral abscess (especially in women), may also be responsible for pyuria These conditions can be discovered by the urethroscope.

(*b*) **Cystitis:** If the urine is alkaline (frequently of offensive odor), and the pus, which is often gelatinous and ropy, issues with the last portion of the urine, then the pyuria is due to cystitis The cystoscope may be employed to confirm the diagnosis, and the urethral sound to discover a possible calculus

(*c*) ***Ureteritis:*** The cause of a pyuria when due to tuberculosis or gonorrheal stricture at the lower, middle or upper part of the ureter, may be determined only by ureteral catheterization Often it is difficult to differentiate between ureteritis and pyelitis

(*d*) **Pyelitis or Pyelonephritis:** The continuous or remittent presence of pus in an acid urine suggests tuberculosis, calculus, or obstructive pyelitis.

[1] Butler Diagnostics in Internal Medicine, D Appleton & Co.

is lost on standing; this change is slower in a dark and cool place where the solution may be kept fresh for some days. Though this reaction is given by acetone, it is so much more sensitive to acetoacetic acid, and the latter so predominates over acetone in fresh urine that the test should properly be regarded as one for acetoacetic acid. It is valuable as being the most delicate indication of acidosis, and one purpose in treatment is, therefore, to keep this reaction continuously negative As it is sensitive to one part of acetoacetic acid in 20,000, while ferric chloride reacts only to about one part in 8000 and in urine is often *indistinct in its fainter degrees, comparison* of different tests is useful. If the nitroprusside reaction is heavy and the ferric chloride negative or faint, there is only slight ketonuria

Vanillin Test for Acetone (Leffmann and Trumper) [1] "About 5 cc. of the urine to be tested is mixed with a strong alcoholic solution of vanillin in a test tube and a fragment of sodium hydroxide dropped in Acetone is promptly shown by a red ring which deepens *rapidly and persists for a long time.* The Rothera-test color fades out soon and is further found to be unsatisfactory in the presence of albumin (the albumin is thrown out of solution when the urine is saturated with ammonium sulfate), but the vanillin test is still quite satisfactory under this condition."

Acetonuria may be found in diabetes mellitus, inanition, septicemia, and grave febrile conditions (typhoid fever, pneumonia, etc). It is also observed in cancerous cachexia.

In the treatment of diabetes mellitus, when acetone is persistently present in large amounts, recourse should be had to the use of insulin (Iletin), as this *remedy judiciously employed will clear* the urine of this product.

Hematuria (Blood in the Urine)

Hematuria is the result of a lesion somewhere in the urinary tract. The quantity of blood may be large or small It may be the result of a lesion in the urethra, prostate, bladder, ureters, or kidneys; or be due to a blood dyscrasia. Bloody urine may also result from contamination during menstruation or the puerperium. When it becomes necessary to examine a woman's urine at such periods, only a catheterized specimen should be used In the presence of hematuria it is important to decide its *origin and cause* The patient is therefore asked at one urination to void urine in three glasses in succession so that the "three-glass test" can be made Also it is important to note the reaction of *the urine, for red cells may hemolyze* in an alkaline urine, so that a hematuria may be mistaken for a hemoglobinuria

Urethral Origin: Blood appears in the first glass and is frequently clotted, *and preceded by a plug of coagulated* blood This may be due to traumatism of the urethra (*e g.*, acute urethritis, after coitus, after passing a sound), or to the presence of foreign bodies, *including stone.*

Prostatic Origin: Blood appears in the first and the third glasses only; this may be due to carcinoma, *calculi or* benign hypertrophy.

Vesical (Bladder) Origin: The blood may be bright red or dark; it may or may not contain small clots; all the urine may be *intimately mixed*

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[1] Leffmann and Trumper: Bull. Wagner Free *Institute* of Science, March, 1926, vol 1, Philadelphia Pa

tent of the urine is very high. The estrin content of nonpregnant urine is between 50 and 100 international units per liter. During the premenstrual period the urine may contain from 150 to 300 units per liter. In amenorrhea, dysmenorrhea and functional sterility the estrin content of the urine is low

Prolan B: The anterior pituitary-like hormone is found in large quantities in the urine of pregnancy (from 25,000 to 100,000 international units to the liter), and in the presence of certain ovarian tumors

Male urine also contains some estrin and in certain testicular tumors there may appear large quantities of estrin or of prolan.

Androsterone: The male hormone appears in various quantities in the urine of males during their fertile stage

Hematoporphyrin

Hematoporphyrin is an iron-free reduction of hematin occurring in small quantities in the blood and is eliminated by the feces and in minute quantities by the urine Large quantities of hematoporphyrin renders the skin sensitive to ultraviolet light and increases cutaneous pigmentation.

Hematoporphyrinuria: An increased amount of porphyrin in the urine imparts to it a Bordeaux red, dark red or port wine color Hematoporphyrinuria is found in conditions causing an increase of hematoporphyrin in the blood, bones, teeth and serous effusions It is also found in lead poisoning, hematochromatosis, cirrhosis of the liver, degenerative lesions of the liver, in tuberculosis, rheumatic fever, pneumonia and other infectious diseases Congenital hematoporphyrinuria is found among those who have other congenital metabolic disturbances.

Diazo Reaction

Urochromogen appears only in abnormal urine and will give a positive reaction with permanganate. The urochromogen reaction is usually positive in such urines as yield a positive diazo reaction.

A positive diazo reaction constitutes the production of a red color in the urine when treated with Ehrlich's diazo reagent.

The diazo reaction was formerly considered an important diagnostic procedure in the diagnosis of typhoid fever. A positive diazo reaction in the urine is obtainable in the following conditions: Typhoid fever, from the middle of the first to the third week; its reappearance after the third week indicates relapse; in measles during the early stages; and in tuberculosis. It may also occur in typhus fever, scarlet fever, erysipelas, rheumatic fever and pneumonia, and less frequently in diphtheria, leukemia, heart failure, carcinoma of the stomach and cirrhosis of the liver. A positive diazo reaction of the urine may at times be obtained after the administration of large doses of quinine, cinchophen, quinidine, salicylates, phenol, creosote, naphthalene, morphine and other opiates, and menthol.

Microscopic Examination of the Urine

After having made a physical and chemical study of the urine a microscopic study completes the examination; preferably a centrifuged specimen is examined which may reveal the presence of the following:

If the pus flows intermittently it is more likely to be caused by suppurative or surgical kidney, with abscesses of considerable size. A coexisting cystitis causes the urine to assume the cystitic type, and also suggests the possibility of an ascending renal infection. Ureteral catheterization may determine beyond doubt the presence or absence of pyelitis.

(*e*) **Outside Sources of Pyuria:** Certain suppurative foci may rupture into the urinary tract (almost invariably into the bladder), usually due to salpingitis, simple or tuberculous, but also arising from an abscess of the ovary or extrauterine pregnancy, suppurating ovarian or dermoid cyst, and psoas or acetabular abscess connected with disease of the vertebrae, or hip joint. A vesicointestinal fistula, or malignant disease involving the bladder by contiguity, may also be classed under this head.

A bacteriological examination of the pus or a culture of the urine may afford valuable evidence by revealing the gonococcus, the tubercle bacillus, colon bacillus, or the bacillus of typhoid fever, as well as the ordinary pyogenic organisms.

Bile

Bile pigments and bile acids in the urine are found in obstructive and toxic jaundice but not in hemolytic or acholuric jaundice. When the bile concentration in the blood exceeds four parts per 200,000 of serum, bile appears in the urine. The greater the concentration of bile in the blood, the greater is its quantity in the urine.

Test for Bile in the Urine: Shaking of bile containing urine will form a yellowish foam. When a white piece of filter paper is immersed in bile containing urine it will be stained yellowish Bile containing urine is acid in reaction and may give a positive albumin reaction

Urobilin and Urobilinogen

Urobilinogen is found in small amounts (1 to 4 mg. in 24 hours) in normal urine Urobilin does not appear in fresh normal urine. In stale urine its presence is due to converted urobilinogen. Large amounts of urobilin in the urine signify the excessive formation of bilirubin. This is found in obstructive and hepatocellular jaundice, in hepatic cirrhosis, in congestion, in gallstones, and in pernicious anemia due to liver insufficiency or to hemolysis. In liver insufficiency the liver cells are incapable of transforming urobilin into bilirubin; and when excessive hemolysis takes place because of blood dyscrasias the liver, though normal, is unable to store the large amounts of urobilin thus formed. The excess of urobilin and urobilinogen is eliminated by the urine. Urobilinuria is therefore an indication of liver damage or of hemolysis.

Test for Urobilinogen: A few drops of Ehrlich's aldehyde reagent added to the urine will give a red color in the presence of urobilinogen.

A total absence of urobilinogen from the urine indicates complete obstruction of the bile ducts.

Hormones in the Urine

Estrin: The estrin content of female urine varies in relation to menstruation During the first few days following menstruation the estrin content of the urine is very low; several days preceding the flow the estrin content is fairly high. During pregnancy and in certain tumors of the ovary, uterus, adrenals and pituitary the estrin con-

acetic acid. They are commonest in pyelonephritis.

(7) **Waxy Casts:** Yellow, highly refractive casts, with clean-cut contours, and often exhibiting irregular curves, notches and fractures; rare except in severer forms of chronic renal disease.

(8) **Fatty Casts:** Made up of masses of fat droplets, often arranged in groups corresponding to renal epithelial cells They are probably remains of true epithelial casts.

Miscellaneous Constituents of Urine

Tissue Fragments: Bits of mucous membrane may be desquamated and passed with the urine (acute cystitis) Fragments of a papilloma or of a carcinoma may be found, and studied histologically.

Spermatozoa: These are present in normal urine after coitus or onanism. They may also be present in the different forms of spermatorrhea. Their form is characteristic, so that they are easily identified.

Animal Parasites: In temperate climates it is rare to find animal parasites in the urine, but they are much more common in the tropics. Among them may be mentioned: (*a*) Amebae; (*b*) echinococcus (hooks, membranes); (*c*) filarial larvae (tropical hematuria and chyluria); (*d*) eggs of the human blood fluke, *Schistosoma haematobium* (bilharziasis or Egyptian hematuria); (*e*) oxyuris or pinworm, occasionally (in young girls), wanders through the urethra into the bladder, (*f*) *Trichomonas vaginalis* (of no import).

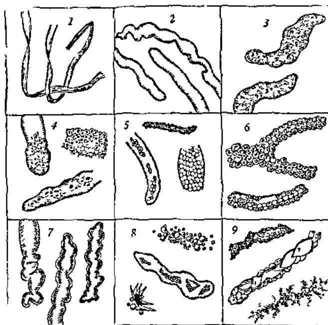


Fig 1—Principal varieties of urinary tubecasts· 1, Cylindroids; 2, hyaline casts; 3, granular casts, 4, epithelial casts, 5, blood casts, 6, pus casts; 7, waxy casts, 8, fatty casts, 9, pseudo-casts (after Grimbert)

Epithelial Cells

Cells from the *tubules of the kidney* are round and about one-third larger than pus cells.

Those from the *pelvis of the kidney* are twice the size of a pus cell and cuboidal or pear-shaped.

Those from the *ureter* are round and slightly smaller than those from the pelvis.

Cells from the *bladder* are flat and square; these are the largest cells encountered, with the exception of those from the vagina.

Cells from the *urethra* are smaller than those from the bladder; they may be cuboidal or columnar. All epithelial cells are granular and contain a relatively small nucleus.

Red Blood Cells

These are due to hemorrhage some where in the genitourinary tract

Casts or Urinary Cylinders[1]

Tube casts are masses of material, deriving their cylindrical shape from the urinary tubules in which they are molded. They are present in the urine in most nephropathies, being most num erous in the acute nephropathies and in the chronic nephropathies with renal edema, less numerous in those asso ciated with contraction of the kidneys They are also present in the urine ir chronic passive congestion (stasis kid ney), in febrile albuminuria, and in jaundice (stained yellow). In acidosis with threatened diabetic coma, showers of short granular casts (coma casts), may appear. Showers of hyaline and, sometimes, of granular casts occur in exacerbations of renal disease.

[1] Barker: *Monographic Medicine, 1916*, D. Appleton & Co.

Several varieties of *casts* occur: (1) Cylindroids; (2) hyaline; (3) granular; (4) epithelial; (5) blood; (6) pus; (7) waxy; (8) fatty casts, etc.

(1) **Cylindroids:** Mucous threads, often twisted and curled, resemble hyaline casts, but are not true casts. They often occur in mild renal disturbance due to passive congestions.

(2) **Hyaline Casts:** Pale, transparent, homogeneous casts, with delicate contours and rounded ends (often hard to make out). The commonest form of cast, indicating the existence of a nephropathy, but throwing no light on the variety of nephropathy. Some of the so-called cylindroids are probably hyaline casts with pointed ends, while others are false casts, composed of mucus

(3) **Granular Casts:** Similar to (2) but the substance is finely granular, usually rather short and plump, often yellowish. The granules may be coarse or fine; they are soluble in acetic acid. One sees various transitions to epithelial casts Granular casts are met with chiefly in the inflammatory and degenerative nephropathies.

(4) **Epithelial Casts:** Aggregations of renal epithelium, sometimes preserving their original arrangement in the tubules (epithelial tubes). The cells are often filled with granules or fat droplets, or there may be a homogeneous necrosis. We distinguish these casts consisting of epithelium from the hyaline and granular casts that have a few epithelial cells upon their surface.

(5) **Blood Cell Casts:** Red cells in masses, molded by the renal tubules The blood comes from the glomeruli (hemorrhagic glomerulonephritis).

(6) **Pus Casts** (leukocyte casts). Cellular casts; the single cells are seen to have polymorphous nuclei on adding

acetic acid. They are commonest in pyelonephritis.

(7) **Waxy Casts:** Yellow, highly refractive casts, with clean-cut contours, and often exhibiting irregular curves, notches and fractures; rare except in severer forms of chronic renal disease.

(8) **Fatty Casts:** Made up of masses of fat droplets, often arranged in groups corresponding to renal epithelial cells They are probably remains of true epithelial casts

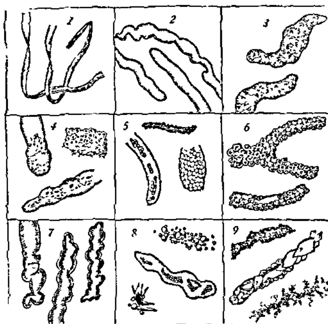


Fig. 1—*Principal varieties of urinary tubecasts:* 1, Cylindroids; 2, hyaline casts; 3, granular casts; 4, epithelial casts; 5, blood casts, 6, pus casts; 7, waxy casts, 8, fatty casts; 9, pseudo-casts (after Grimbert).

Miscellaneous Constituents of Urine

Tissue Fragments: Bits of mucous membrane may be desquamated and passed with the urine (acute cystitis) Fragments of a papilloma or of a carcinoma may be found, and studied histologically.

Spermatozoa: These are present in normal urine after coitus or onanism. They may also be present in the different forms of spermatorrhea. Their form is characteristic, so that they are easily identified.

Animal Parasites: In temperate climates it is rare to find animal parasites in the urine, but they are much more common in the tropics. Among them may be mentioned: (*a*) Amebae; (*b*) echinococcus (hooks, membranes); (*c*) filarial larvae (tropical hematuria and chyluria); (*d*) eggs of the human blood fluke, *Schistosoma haematobium* (bilharziasis or Egyptian hematuria); (*e*) oxyuris or pinworm, occasionally (in young girls), wanders through the urethra into the bladder; (*f*) *Trichomonas vaginalis* (of no import).

Vegetable Parasites and Bacteria: These are of no importance when seen in urine, unless they are found in a specimen obtained by aseptic catheterization.

In *bacteriuria,* the urine is usually turbid, especially if the bacteria are motile. It may be impossible to make such urines clear by centrifugalization The bacteria may be studied by microscopic examination (fresh drop, smear), by cultural methods, or by animal inoculation.

Among the nonpathogenic bacteria that may be present are: (*a*) Micrococcus urae; (*b*) bacterium urae; (*c*) urinary sarcina; (*d*) several nonpathogenic streptococci; (*e*) bacillus cystiformis (Clado); (*f*) bacillus proteus

Pathogenic Bacteria: The finding of the *tubercle bacillus* in the urine is of the greatest clinical significance. It occurs both in cases of generalized tuberculosis (as a result of bacillemia), and more particularly in cases of tuberculosis of the genitourinary organs. In this last condition it is usually associated with a pyuria, and frequently with a hematuria.

In searching for tubercle bacilli in the urine it is of especial importance to obtain an uncontaminated specimen since the smegma bacillus may readily lead to confusion. The sediment from about 50 cc. of thoroughly centrifugalized urine should be used. If much pus is present antiformin treatment of the sediment may be advisable. In all doubtful cases resort should be had to guinea-pig inoculation.

Gonococcus is of great diagnostic importance. This is an intracellular, biscuit-shaped diplococcus best seen in smears stained with methylene blue. It decolorizes by the Gram method.

Bacillus coli is of considerable diagnostic importance (cystitis; pyelitis).

Bacillus typhosus is of importance for prophylaxis (bacillus carrier), and also for diagnosis in the rare cases of pyonephrosis due to the typhoid bacillus Throughout the course of typhoid fever, after the first week, typhoid bacilli are often demonstrable in the urine.

Pyogenic cocci are rare as a cause of cystitis and pyelitis. *Streptococci* are not uncommon in acute nephritis. Staphylococci are seen occasionally in general sepsis (adolescence).

Artifacts: Urinary sediments may be accidentally contaminated by foreign bodies of various sorts, *i. e.,* starch granules, cotton fibers, linen fibers, silk fibers, wool, fat globules, etc

Fat: Fat in the urine appears as globules Normally, fat may appear in the urine (*lipuria*) following the administration of large quantities of oil or a high fat diet Pathologically, fat in the urine may be due to diabetes mellitus, lipoid nephrosis, fracture of bone with injury to the bone marrow. It may also follow maceration or injury to the superficial fat. Lipuria may also occur in alcohol and phosphorus poisoning and in pyelitis, pyonephrosis and nephrosis

Crystalline Deposits (After Faught)[1]

Acid Group: ***Uric Acid:*** These crystals are yellow, reddish brown or brown in color. The most characteristic forms are rhombic prisms or lozenge-shaped crystals. These may occur singly, but more often they are united in irregular masses.

Urates: The urates, chiefly the urate of sodium and potassium, if they do not

[1] Faught: Essentials of Laboratory Diagnosis. 1921, F. A. Davis Co.

appear as an amorphous deposit, show as crystals in the forms of needles or dumbbells, of reddish brown color, and also in globular masses which are dark brown and almost opaque, with or without projecting spines.

Oxalates: The usual form of calcium oxalate in the urine is a perfect octahedron without color. More rarely they appear in the conventional hourglass form. This form is somewhat similar to the urate, from which it may be distinguished by the total absence of color in the oxalates

Carbonates: These are rare, but if present evolve bubbles of gas when treated with hydrochloric acid under the microscope.

Sulfates: This is a rare form of deposit which, when present, appears as fine feathery crystals. Frequently a number of crystals radiate from a common center.

Alkaline Group: ***Phosphates:*** These may occur as a semiopaque amorphous deposit without color. More commonly they appear as the characteristic coffin-lid crystals. A less common form of crystalline phosphatic deposit appears as fine, branching, feathery crystals, which have been likened to the needles and branches of the pine tree.

Ammonium Urate: These are characteristic of the uric acid and urate group in that they are yellow or brownish in color. In alkaline urine the urates appear as fine feathery spheres of varying size, somewhat resembling chestnut burrs.

Cholesterine: This is a rare form of deposit which appears as irregular flat platelets whose sides follow the characteristic lines of a parallelogram, the angles of which are often irregular Not infrequently the platelets are seen in overlapping groups

Cystine: This is a rare deposit. When present it appears as irregular transparent plates of varying size often in overlapping groups

CHAPTER XXXIV

Blood Examination

Normal Blood Findings

(PHYSICALLY, CHEMICALLY AND BIOLOGICALLY NORMAL)

1 *Quantitative Relation* 40 60 to 45 55
2. *Color* Bright red for arterial and dark red for venous blood.
3 *Hemoglobin* 90 to 100 per cent in men and 85 to 90 per cent in women—16 to 17 Gm. per 100 cc. in men and 15 to 16 Gm in women.
4. *Reaction* pH 7.35 to 7 39. (See Graph by Trumper, p 1012)
5 *Specific Gravity* 1 045 to 1 075
6 *Bleeding Time* One to three minutes
7. *Coagulation Time* Four to five and a half minutes—should not exceed ten minutes
8. *Retraction of Clot* One to two hours, and is complete in from 18 to 24 hours.
9 *Sedimentation Rate* 9 mm. for men and 12 mm. for women when blood column is 50 mm. high
10. *Red Blood Cells* 4,500,000 to 5,500,000 per cm for men and slightly less for women
11 *Saturation Index* 0 87 to 1.23
12. *Color Index* 0 85 to 1.15
13. *Volume Index* 0 99 to 1 02
14 *Leukocytes (white blood cells) per cm* 5000 to 10,000.
15. *Myelocytes*. Occasional
16. *Juveniles* 8 to 16 per cent.
17. *Neutrophils* 60 to 75 per cent.
18 *Eosinophils* 1 to 4 per cent
19 *Basophils* Occasional.
20. *Monocytes* 2 to 6 per cent.
21 *Large Lymphocytes* 2 to 4 per cent
22 *Small Lymphocytes*· 15 to 35 per cent.
23. *Thrombocytes (Platelets)*· 250,000 to 350,000
24 *Reticulocytes* 1 per cent.
25. *Abnormal Cells:* Occasional.
26. *Total Solids:* 19 to 23 mg. per 100 cc.
27. *Total Acetone Bodies*· 1.3 to 2.6
28. *Serum Amylase:* 70 to 200 units.
29. *Prothrombin Time:* 10 to 20 seconds.
30. *Creatine*· 3 to 7 mg. to 100 cc. of blood.
31. *Total Nonprotein Nitrogen*· 25 to 35 mg. to 100 cc. of blood.
32 *Urea Nitrogen* 12 to 15 mg. to 100 cc. of blood.
33. *Creatinine* 1 to 2 mg. to 100 cc. of blood.
34. *Uric Acid* 2 to 3.5 mg. to 100 cc of blood.
35. *Glucose* 80 to 120 mg to 100 cc. of blood.
36 *Calcium:* 9 to 11 mg to 100 cc of blood.
37 *Chlorides* 400 to 500 mg. to 100 cc. of whole blood; 570 to 620 mg. to 100 cc. of plasma
38 *Total Proteins*. 6 5 to 8.2 per cent.
39 *Albumin* 4 6 to 6 7 per cent.
40 *Globulin* 1 5 to 2 5 per cent.
41. *Iodine*· 8 to 16 gamma or micrograms (or 0.008 to 0 016 mg.) per 100 cc. of blood
42 *Cholesterol* 140 to 200 mg per 100 cc. of blood serum.
43 *Cholesterol Esters* 60 to 80 per cent of the total cholesterol.
44 *Free Cholesterol* 20 to 40 per cent of total cholesterol.
45 *Phosphorus* (Inorganic) · 3 5 to 4 mg. per 100 cc. of blood in adults 5 to 6 mg per 100 cc of blood in children. Phosphorus (Lipid): 2 5 to 14 5 mg per 100 cc of blood serum
46 *Phosphatase* Two to four Bodansky units (0 10 to 0 21 Kay units) Plasma phosphatase is 0 15 mg. per 100 cc of blood, higher values in growing children The figure 0 15 has reference to inorganic phosphates converted from sodium glycerophosphate in 48 hours at 38° C and pH 7.6 by the action of 1 cc. of plasma.
47. *Bile* One part of pigment to 500,000 of serum (0.1 to 0 8 as bilirubin)
48. *Icterus Index* (color of serum compared with a 1 to 10,000 solution of potassium dichromate representing an icterus index of one): Four to six per cent.
49 *Van den Bergh Reaction* 0.2 to 0 8 per cent.
50 *Red Cell Fragility:* Hemolysis begins with 0 45 NaCl and is completed with 0.35 NaCl solution.
51 *Alkali Reserve* 77 to 53 volume per cent; ten per cent lower in children.

52. *Blood Volume:* Five to six liters or about 75 cc. per kilogram of body weight, or approximately 1/11 of the body weight Somewhat lower in children
53. *Fat, Total* 400 to 1400 mg.; neutral 0 to 370 mg, fatty acids 290 to 450 mg.
54. *Potassium:* 16 to 22 mg per 100 cc. of blood serum.
55. *Sodium* 315 to 340 mg. per 100 cc. of blood serum
56. *CO_2 Capacity* 55 to 80 volume per cent; CO_2 content of arterial blood, 45 to 55 volume per cent; CO_2 content of venous blood, 50 to 60 volume per cent
57. *Fibrinogen* 0 2 to 0 4 mg per 100 cc of blood serum.
58. *Magnesium* 1 8 to 3 6 mg per 100 cc of blood serum.
59. *Cevitamic Acid* 0 6 to 2 5 mg per 100 cc of blood serum (vitamin C)
60. *Total Base* (milliequivalents per liter) 155
61. *Iron* 52 mg per 100 cc of blood.
62. *Lactic Acid* 6 to 20 mg per 100 cc of blood
63. *Serum Volume* 49 to 59 per kilogram of body weight

Definition of Terms Employed in Hematology

Anemia: The red cells and hemoglobin are chiefly affected (Diminished in number and quantity)

Leukemia: Changes in the leukocytes are chiefly observed (Increased in number)

Plethora: An abnormal increase in the total *quantity* of blood

Anhydremia: A diminution in the normal quantity of fluids in the blood.

Oligochromemia: An abnormal diminution in the amount of hemoglobin

Oligocythemia: A diminution in the number of red blood cells

Polycythemia: An increase in the number of red blood cells (erythrocytosis)

Leukocytosis: An abnormal increase in the number of white cells.

Leukopenia: An abnormal decrease in the number of white cells

Microblasts: Small nucleated red *blood cells.*

Normoblasts, Erythroblasts: Nucleated red blood cells (of normal size).

Megaloblasts: Large nucleated red blood cells.

Macrocytes: Large red blood cells (nonnucleated).

Microcytes: Small nonnucleated red blood cells.

Megalocytes: Same as macrocytes or gigantocytes.

Reticulocytes: Immature erythrocytes containing a threadlike reticulum, stainable with vital stains.

Erythrocytes: Red blood cells of normal size (nonnucleated).

Poikilocytes: Irregularly shaped red blood cells

Anisocytosis: Excessive variation in the size of the red corpuscles

Polychromatophilic Degeneration (Ehrlich) · An atypical staining reaction *of the erythrocytes.*

Basophilic Granulation (stippling) A peculiar granular degeneration of the red blood cells (characteristic in lead poisoning, malaria, and in severe anemia).

Howell-Jolly bodies are granules found in red cells; they are stainable with basic stains.

Cabot's bodies are probably nuclear remains appearing as intra- and extracellular rings which stain with acid dyes

Hemanalyses (Blood Examinations)

Blood examinations comprise.

Blood Count: Hemoglobin determination, number and kind of red cells, white cells and platelets.

Blood chemistry for glucose and other constituents of the plasma.

Serologic Tests: Blood cultures, complement fixation tests, etc.

millimeter. When it becomes necessary to examine the blood corpuscles more carefully, in order to ascertain the characteristics of the red cells and the variety of the whites, a film of blood on a slide stained with Wright's stain is examined. The examination of the blood by stained specimen is usually known as the *differential count.* It is extremely important in many instances to have a differential count made, because various blood diseases and inflammatory conditions may be recognized by this means. In the normal blood the differential count shows as follows:

Red Corpuscles (erythrocytes), about 4,500,000 to 5,500,000 to 1 cmm. of blood.

White Blood Cells (leukocytes), 5000 to 10,000 in 1 cmm of blood.

Polymorphonuclears: 65 to 70 per cent.

Small Mononuclears: 20 to 30 per cent.

Large Mononuclears: Four to eight per cent.

Transitionals: One to three per cent.

Eosinophils: One to four per cent.

Basophils (mast cells): One-quarter to one-half per cent (occur only occasionally).

Platelets: Approximately 300,000 per cmm.

Hemokonia (blood dust).

Reticulocytes: One-half to one per cent.

Significance of Abnormal Blood Counts

Hemoglobin

The amount of hemoglobin, whether calculated on a percentage basis or in grams, is important only in relation to the number of red corpuscles which is considered as the color index. Normally the color index is 1 or somewhat lower.

An increased color index is found in pernicious anemia, during crisis of hemolytic jaundice, in sprue, and occasionally in carcinoma of the intestine, pellagra and other conditions that cause a *hyperchromic macrocytic anemia.*

A *decreased color index* is found in chlorosis and in many of the secondary anemias, particularly of the hypochromic microcytic type; also in polycythemia vera.

An *actual increase** in the amount of hemoglobin and not an increase in relation to the number of erythrocytes is found in polycythemia vera. in cyanosis due to congenital heart disease, and in chronic pulmonary disease, such as asphyxia, and anhydremia. An *actual* asthma, emphysema, pulmonary stenosis, *decrease* in the amount of hemoglobin is found in all types of anemia.

Red Cells

An increase in the number of erythrocytes is found in polycythemia vera, Ayerza's disease, hemoconcentration, shock, dehydration and in high altitudes

A decrease in the number of red cells is found in all types of anemia, whether primary or secondary. A very low red corpuscle count is found in pernicious anemia, in aplastic anemia, after severe hemorrhage and in hemolytic jaundice.

Differential Red Cell Count: *Normoblasts* are found in severe types of anemia such as pernicious anemia, chlorosis, and in the advanced stages of most of the anemias. Their presence in the blood stream indicates increased marrow activity and nature's attempt to replenish the circulation with red cells that are being rapidly destroyed. Normoblasts are not found in aplastic anemia

Megaloblasts are found in pernicious anemia, *in other hyperchromic types of* anemia and in myelocytic leukemia. These cells, because of their nuclei, may resemble monocytes.

Microcytes are found in the iron deficiency anemias such as chlorosis and in various types of secondary anemia presenting a low color index These cells are often extremely irregular in shape.

Macrocytes are found in pernicious anemia and in the various anemias associated with a high color index. Macrocytes often appear as large oval-shaped cells.

Sickle-shaped red cells are found in sickle cell anemia.

Oval or *elliptoid red cells* (ovalocytes) occur as a familial peculiarity and may not be associated with disease.

Poikilocytes are irregularly distorted cells. They occur in most of the severe anemias, usually in association with *anisocytes* (irregularly sized cells).

Reticulocytes (reticulated immature red blood cells) Erythrocytogenic hyperactivity of the bone marrow is marked by the appearance of an increased number of reticulocytes in the peripheral blood stream These cells are found in large numbers in the blood of normal newborn babies, also in some of the anemias where there is increased bone marrow activity (hyperplasia), and in hemolytic jaundice An increase in the reticulocyte count in a patient with pernicious or other types of anemia when under treatment indicates a favorable response. When the bone marrow is aplastic, the reticulocytes are absent from the blood stream and fail to appear under treatment.

Polychromatophilia (varied colored red cells) are found in severe anemias and leukemia; their presence in the blood stream indicates an increased regeneration of red cells. These cells are in the embryonic state and do not stain readily with acid stains and but poorly with basic stains. Wright's stain colors then, light blue or a dirty blue-red

Granular basophilic degeneration (stippling) of red cells indicates abnormal regeneration of erythrocytes. These cells are recognized by the appearance of blue granules on a dirty blue or brownish background when stained with Wright's stain. Stippling is found in lead poisoning, pernicious anemia, leukemia and in severe secondary anemia, particularly of toxic origin.

Achromia are colorless red cells or, rather, red cells that show a large central pale depression surrounded by a narrow pink margin are an indication of a low hemoglobin content.

Howell-Jolly bodies are found in the red cells of pernicious anemia, hemolytic icteroanemia, leukemia, in severe types of secondary anemia and after splenectomy.

Cabot's ring bodies are found in severe anemias and in lead poisoning.

Fragility of Erythrocytes. (Resistance of erythrocytes to hemolysis) Normal fragility is 0 45 to 0 34 per cent.

The fragility is increased (resistance decreased) in hemolytic jaundice, hemolytic icteroanemia, and sickle cell anemia It is decreased (resistance increased) in obstructive jaundice, aplastic anemia, pernicious anemia, lead poisoning and after splenectomy.

Sedimentation of Red Cells and the Blood Sedimentation Test

In health the erythrocytes in a citrated specimen of blood settle towards the bottom of a vessel within a fairly definite period. In certain diseases and

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under certain circumstances the settling down or the sedimentation rate of the red cells are delayed. The rapidity of *the sedimentation also depends upon the* plasma stability and the number and size of the red cells.

The blood sedimentation test depends upon the length of time it requires for the red corpuscles in a given quantity of citrated blood to settle downwards in its serum. The sedimentation test consist of the measuring of the speed with which the red corpuscles separate from the plasma of noncoagulating blood. It has been observed that the erythrocytes settle perceptibly slower in health than in disease, and that the graver the disease, the more rapidly will the red corpuscles settle in the blood serum. It may, therefore, be stated that the sedimentation time is longer in health than in disease, and that it is comparatively short in grave illness. In normal adult men the sedimentation time is longer than in women, and is also longer in the newborn and the aged.

There are various methods and modifications in use for determining this test. The three most important methods are:

1. **The Distance Method of Fahraeus** (*modified by Westergreen*): This consists in measuring the distance the red corpuscles in a definite quantity of citrated blood (in a standard tube) have settled at the end of one hour, two hours and 24 hours.

Technic: One part of 3 8 per cent of sodium citrate solution is mixed with four parts of blood and gently agitated This mixture is poured up to the 200 mark into a graded glass pipette tube, measuring 300 mm. in height and 2.5 mm. in diameter. The tube is stood upright so that the erythrocytes may settle; after one hour, two hours and 24 hours, the height of the column of supranatant fluid is measured so as to determine the level attained by the red corpuscles during 24 hours. In healthy men, the supernatant fluid column after one hour measures 3 mm. and in healthy women it measures 5 to 10 mm. By the end of 24 hours, the entire quantity of red blood cells should be settled toward the bottom of the tube.

2. **The Time Method of Linzenmeier:** This consists in determining the length of time required for the red corpuscles to settle to a definite level in a standardized glass tube.

Technic: Citrated blood (of the same dilution as used in Method 1) is poured into a glass tube measuring 6 5 cm. in height and 5 mm. in diameter, and is marked at two levels—the upper level at 1 cc. and the lower level 18 mm below the first mark. The quantity of blood should be sufficient to reach the level of the 1 cc. mark. The tube is then allowed to stand upright and the length of time required for the corpuscles to settle from the 1 cc. mark to the 18 mm. mark (the sedimentation time) is noted The normal sedimentation time for healthy men is from 20 to 23 hours, and for healthy women—from 13 to 16 hours. During menstruation it may be as low as 10 hours. Sedimentation time of less than three and one-half hours is considered as pathologic.

3. **The Graphic Method of Cutler:**[1] This blood sedimentation test is practically a combination of the Distance and Time Methods and is superior to either method alone, because the velocity with which the erythrocytes settle varies at

[1] Cutler, Jacob: J. A. M A. 182, 6, clxxi, June, 1926

Cutler's Blood Sedimentation Test Chart.[1]

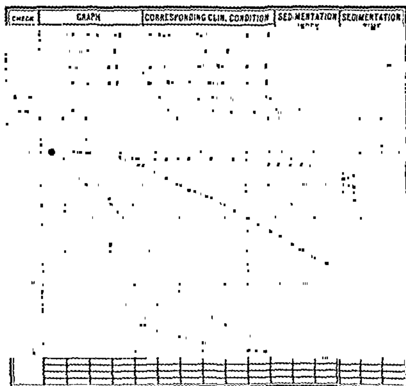


Fig 1

The graphs are actual reproductions of the sedimentation phenomenon.

——— × ——— × ——— Horizontal line (clinically healthy individual).
——— ● ——— ● ——— Diagonal line (clinically quiescent tuberculosis).
——— O ——— O ——— Diagonal curve (clinically slightly active tuberculosis).
——— ×× ——— ×× ———Vertical curve (clinically markedly active tuberculosis).

Explanatory Notes. Sedimentation Index. Represents the total sedimentation of red blood cells *at the end of 60 minutes expressed in millimeters Normal index* for men varies from 2 to 8 millimeters, with an average of 3 to 4; for healthy women from 2 to 10 with an average of 5 to 6

Sedimentation Time Represents the time required for the "complete" settling of the red blood cells Normal time is always a question of hours. Of clinical value when reduced to 60 minutes or less.

Horizontal Line. A straight line with a sedimentation index falling within normal limits It also represents normal The other graphs are always abnormal findings.

Diagonal Line A straight line with a sedimentation index beyond normal limits

Diagonal Curve. A curve of gradual descent with a sedimentation index beyond normal limits and a sedimentation time of 35 to 60 minutes

Vertical Curve A curve of sharp descent with a sedimentation index beyond normal limits and a sedimentation time of 30 minutes or less.

[1] Cutler, J · The Graphic Presentation of the Blood Sedimentation Test, Am. J. Med. Sc, 171 · 882, June, 1926.

certain times and this variation can be recorded by the graphic method.

Technic: A special test tube adequately marked is necessary. The tube devised by Cutler is of 5 cc. capacity graduated into tenths of a cc. each 1 mm. in height and marked in mm

Four and a half cc. of fresh blood is mixed with a half cc of a 3 per cent citrate solution and poured into the test tube. The mixture is gently agitated and the tube is stoppered and the reading *is done every* five minutes for an hour by noting boundary zone between the erythrocytes and the plasma. The observations are recorded on the sedimentation charts on which the horizontal lines represent the divisions on the sedimentation tube and the vertical lines, the intervals of time In this way, a graph is traced which shows the position of the sedimenting column of red blood cells at any period of time during the first hour.

The sedimentation value is determined according to the path traversed by the red blood cells during the first hour and depends upon the nature of the graph, the sedimentation index and the sedimentation time Together they furnish all the information that is likely to be obtained from the sedimentation test. The graph serves as a rough estimation of the presence or absence of pathologic activity. The sedimentation index and sedimentation time help to determine the degree.

Sedimentation Index: The normal sedimentation time for men is from 2 to 9 mm. per hour, and for women 2 to 12 mm.

Increased sedimentation rate occurs normally during menstruation and pregnancy. Pathologically, it occurs in most of the infectious diseases during their active stage; in malignant neoplasms; after operations, in wounds, and fractures; in diabetes mellitus; in obstructive jaundice; in salpingitis; in late appendicitis, in tuberculosis; in rheumatic fever, in pregnancy; also after intravenous injections of foreign proteins and of arsphenamine, and after irradiation.

Decreased sedimentation rate occurs in dehydration, hyperprotonemia, polycythemia, rickets, cardiac failure and diseases of the liver associated with jaundice.

The sedimentation time remains normal in nervous and mental diseases, asthma, hay fever, benign growths, peptic ulcer, catarrhal appendicitis, essential hypertension, chronic valvular disease (in the absence of rheumatic fever), and in diseased tonsils or sinuses.

In healthy individuals the volume of red blood cells after complete sedimentation is about 50 per cent of the total volume of blood. In anemia the volume of red blood cells is naturally reduced This is reflected in the sedimentation index. When the sedimentation index is unusually high, regardless of the character of the graph, it indicates among other things a small volume of the red blood cells and should always suggest anemia. In this respect the sedimentation index serves the purpose of the hematocrit

The Leukocytes

The Diagnostic Value of the Leukocyte Count: The number of leukocytes per cubic millimeter of blood in healthy individuals has a normal range from 5000 to 10,000. Under certain conditions in normal subjects, the leukocyte count may be somewhat lower than the low figure or somewhat higher than the high figure. In pathologic conditions there may be a marked reduc-

tion in the number of leukocytes (leukopenia) or a great increase in the number of leukocytes (leukocytosis). It is important to determine not only the total number of leukocytes per mm. of blood, but also the number or percentage, that is, the relative proportion of the various types of leukocytes Thus in the presence of a moderate leukocytosis or a moderate leukopenia, if the neutrophils, monocytes, eosinophils, lymphocytes and other white cells bear a normal proportion to each other, the increase or decrease in the total number of leukocytes bear no specific significance other than a general leukocytic disturbance

Leukocytosis: A white cell count above 10,000 is generally considered as leukocytosis In severe leukocytosis the count may be above 50,000. It should be emphasized that leukocytosis differs from leukemia The former is only a symptom while the latter is a distinct pathologic entity.

Leukocytosis may be physiologic or pathologic.

Physiologic Leukocytosis: The number of leukocytes are seldom high and the differential count is usually normal. Physiologic leukocytosis occurs in the newborn, during menstruation, during pregnancy, during labor, after physical and mental exertion, after a cold bath, after massage, and after taking certain drugs and foods.

Pathologic Leukocytosis: Leukocytosis occurs in most infections, infectious diseases and inflammations (the exceptions to this rule are noted under leukopenia), after paroxysms of tachycardia, in coronary thrombosis, in uremia, during hemorrhage, particularly when bleeding occurs in one of the serous sacs such as the pleura, pericardium, peritoneum, in the joints, subdural and subarachnoid spaces. Severe leukocytosis is also found in periarteritis nodosum, in neoplasms with metastasis to the bone marrow, in severe cachexias, in infectious mononucleosis, in the leukemias and in many other diseases.

In mild infections the leukocytosis is but slightly increased above the normal In moderate infections the leukocyte count is moderately high. In *overwhelming* infections the leukocyte count is either *very high*, or *very low,* the latter indicating a failure of the leukopoietic system to respond because of the severe infection or general debility. The *absence of leukocytosis* in diseases where leukocytosis is the rule is a grave prognostic omen. *Per contra,* the presence of leukocytosis in conditions characterized by leukopenia indicates complications, mixed infections or mistaken diagnosis.

Leukopenia: May be defined as the reduction of leukocytes below the usual normal or its maintenance at the low normal level in the presence of febrile diseases. A low leukocyte count is found in certain toxic states, in several of the infectious diseases, in some of the diseases of the blood-making organs, and in some of the intoxications

Infections Characterized by Leukopenia: Typhoid and paratyphoid fevers, undulant fever, malaria, influenza, virus pneumonia, measles, rubella, dengue, yellow fever, smallpox during the early stage, leprosy, mumps, tuberculous meningitis, pellagra, tsutsugamushi disease, pappataci disease, kala-azar, miliary tuberculosis, leishmaniasis, granulocytopenia (agranulocytic angina), and in overwhelming infections

Diseases of the blood-making organs causing leukopenia are pernicious anemia, aplastic anemia, Banti's syndrome,

Gaucher's disease, Von Jaksch's anemia and the aleukemia stage of leukemia.

Toxic agents causing leukopenia are allergic *states, anaphylaxis,* large doses of x-rays or radium, reactions to various drugs, such as the benzol group, aminopyrin group, arsenic, lead, and antimony

Differential Count: The relative proportions in the number of the various types of white cells are disturbed in many diseases and are often of great diagnostic importance. An increase in a particular type of cell in the blood does not always bear a relation to the total number of leukocytes

The Neutrophils (polymorphonuclear, neutrophilic granulocytes): Normally the neutrophils make up from 55 to 75 per cent of the total leukocyte count In pathological conditions they may be increased to above 90 per cent or decreased to less than two per cent It is also important to note not only the total number of neutrophils but also the stage of their development, which is judged by the nuclear morphology. Normally the segmented forms (adult or mature forms) constitute 55 to 60 per cent of the total leukocyte count. The immature forms are the nonsegmented forms (stabs or adult metamyelocytes); they constitute three to five per cent and the young nonsegmented forms (juveniles) young metamyelocytes are one per cent The myeloblasts are normally absent from the blood. An increase of the immature forms or of the abnormal forms in the blood smear indicates severe infection

An increase in the number of neutrophils is found in abscess, active infections (by bacillus coli, meningococci, pneumonococci, pyocyaneus, staphylococci, streptococci, etc.). Neutrophilic leukocytosis is found in the following diseases: Anthrax; appendicitis; chickenpox; cholelithiasis; cholera; convulsions; coronary thrombosis; dehydration in diabetic coma, eclampsia, diphtheria; endocarditis; erysipelas; furunculosis, gonorrhea, intestinal obstruction; lobar pneumonia; measles; meningitis; Oroya fever; otitis media; pellagra; periarteritis nodosa; peritonitis; peritonsillar abscess; pneumococcus pneumonia; pyelitis; rheumatic fever; salpingitis, scarlet fever; septicemia; serum sickness; slowing of the blood stream; smallpox (during the pustular stage); strangulated hernia; tachycardia (paroxysmal); typhoid fever with complications; typhus fever, and in nearly all acute inflammatory infections. It is also found after strenuous exercise and during the active stage of digestion.

A decrease in the number of neutrophils is found in agranulocytic angina, aplastic anemia, arsenic poisoning, benzol poisoning, infectious mononucleosis, hypochromic anemia, kala-azar, lymphatic leukemia, intense radiation, leishmaniasis, malaria, pernicious anemia, paratyphoid, typhoid fever, purpura, whooping cough, and undulant fever

Eosinophils: Normally the eosinophils form between one and four per cent of the total white count (100 to 400 cells per cmm. of blood). *A decrease in the number of eosinophils* occurs in septic and in some infectious conditions, also in aplastic anemia. In infectious disease when the neutrophils are greatly increased in number, the eosinophils may disappear; their return in the peripheral blood is considered by Simon as an indication of recovery.

An *increase* in the number of *eosinophils* is found in: *Normally, in infants;*

and in adults as a familial characteristic and during menstruation. *Pathologically*, in *allergic conditions* such as bronchial asthma, hay fever, migraine, angioneurotic edema and urticaria (*when not due to serum* disease); in *parasitic infestations* by uncinaria, trichinae, echinococci, filaria, bilharzia, and occasionally by amebae, tenia and tapeworms; in *various diseases*, such as scarlet fever, Hodgkin's disease (not constant), Addison's disease, periarteritis nodosa (not constant), chorea, gonorrhea (not constant), measles, rheumatic fever, malaria, active tuberculosis, and during convalescence from pneumonia; in *certain bone diseases and tumor*, as in osteomyelitis, osteomalacia, rickets, osteitis deformans, osteitis fibrosa cystica, sarcoma and metastatic carcinoma and in other tumors, *after ingestion of various foods and drugs* such as raw liver, camphor, pilocarpine, phosphorus and copper, *in various skin diseases*, such as eczema, pemphigus, dermatitis herpetiformis, herpes zoster, scabies and psoriasis; also in some of the *blood dyscrasias*, as in myelocytic leukemia, eosinophilic leukemia, sickle cell anemia, and after *splenectomy*.

Basophils, Myelocytes and Myeloblasts: These are immature white cells belonging to the granular or myelogenic group They are normally found in the bone marrow and only appear in the blood stream in fairly large numbers in myelogenous leukemia, neoplastic metastasis of the bone marrow and in some of the infections where the Schilling index indicates a shift to the left.

The Lymphocytes

The lymphocytes are cells that arise from the lymphoid tissue In adults they form from 20 to 30 per cent of the total white cell count, and in children they may form 50 per cent of the white cells.

Increased number of lymphocytes (lymphocytosis) is found in lymphatic leukemia, infectious mononucleosis (glandular fever), whooping cough, malta fever, influenza, agranulocytic angina, lymphoma, lymphosarcoma, aleukemic lymphadenosis, syphilis, mumps, pernicious anemia, exophthalmic goiter. A relative lymphocytosis is found in typhoid fever, tuberculosis, rickets, psoriasis, and in conditions where the polymorphonuclear leukocytes are decreased The lymphocytes are *decreased* in such infections as show a great increase in the polymorphonuclear leukocytes, *i. e.*, lobar pneumonia, acute appendicitis and similar acute infections.

Monocytes: These cells possess phagocytic action; they form from two to six per cent of the total white cell count. *An increase* in the number of monocytes in the blood is found in infectious mononucleosis, subacute bacterial endocarditis, malaria, undulant fever, dengue, trypanosomiasis, monocytic leukemia, and often in syphilis, typhoid fever, Hodgkin's disease; also in rapidly advancing tuberculosis, in some forms of septicemia, and in tetrachloromethane poisoning.

Arneth Index

By this is meant the division of leukocytes into classes according to their nuclear arrangements It is assumed that very young leukocytes have a single oval, round or bent nucleus, and as the leukocytes become older their nuclei undergo a change in shape, so that instead of a single, simple nucleus in the very young cell, the older cells present nuclei with two, three, four, five or more lobes. The older the cell, the more complex is the shape of its nucleus.

In health the white corpuscles may be divided into five classes, according to the arrangement of their nuclei.

Class I (with no nuclear lobes, but with simple round or bent nucleus) forms five per cent of the neutrophilic leukocytes.

Class II (with two lobes) *forms 35* per cent.

Class III (with three lobes) forms 41 per cent.

Class IV (with four lobes) forms *17* per cent, and

Class V (with five or more lobes) forms two per cent.

An overabundance of simple nucleied white corpuscles in the circulating blood is assumed by Arneth to indicate an increased leukopoietic activity.

When an increase in the number of simple nucleied white cells exists, it is termed a *shift to the left*, and when *white cells containing complex nuclei* are preponderant, it is termed a *shift to the right.*

The Schilling Index

The Schilling differential count is a simplified modification of the Arneth index whereby the neutrophils are classified as immature or nonsegmented, and mature or segmented forms.

The nonsegmented or immature forms are: Myelocytes, juveniles and stabs. (*a*) The myelocyte has a round or oval-shaped, relatively large, vesicular, coarsely granular nucleus, and usually also a nucleolus. Myelocytes are normally found in the bone marrow and are absent from the normal circulating blood.

(*b*) *Juvenile* cells or young metamyelocytes are somewhat older than the myelocytes; each contains a nearly circular or kidney-shaped nucleus, the concave part of which is directed towards the larger amount of cytoplasm. These cells are normally found in the bone marrow *and rarely in the peripheral* blood.

(*c*) Stab cells are older than the juveniles; the nucleus is usually a rod, *band or ribbonlike* structure often twisted into bizarre shapes resembling the letters U, V, S, T Normally they are found, from two to five per cent, in the peripheral blood.

The mature neutrophils are adult polymorphonuclear leukocytes; each cell contains a nucleus that is divided into two, three, four or five unequal segments or lobes, each connected by a narrow filament. The normal blood contains *from 65 to 75 per cent* of neutrophils of which two to five per cent are stab forms or immature neutrophils

The Schilling *theory* is based on two shifts: (1) A regenerative shift of the neutrophils in which there occur juvenile cells and myelocytes This is found in septic diseases. (2) A degenerative shift in which there occur large numbers of stab nuclears, due to defective *neutrophilic leukopoiesis*. This is found in severe infections. Often there occurs a mixture of the two shifts.

The normal hemogram is made up of erythrocytes, granulocytes, *blood* platelets, lymphocytes and monocytes, indicating a physiologic regeneration of the bone marrow, reticuloendothelial and lymphoid systems, with a physiologic destruction of the cells in the various organs and tissues. In disease there may be evidence of increased production of cells (increase in juvenile forms) or evidence of accelerated destruction, that is, degenerative changes.

The part played by the neutrophils in various infections is described thusly by Schilling: "Slight irritations from toxemia cause functional changes only in the leukocytic picture; medium irritations act through the leukopoietic organs; severe irritations act also upon the development of the individual cells, while very severe irritations restrain through paralysis of the central, and destruction of the central and peripheral cells." In most infections the response of the white cells is as follows First the neutrophils, second the monocytes, and last the lymphocytes. These three phases may temporarily shift or the rarer types of cells may appear depending upon the type of infection.

In acute infection with a favorable course, Schilling notes three phases:

(1) "The neutrophilic battle phase" which is characterized by leukocytosis, left nuclear shift, some degenerative nuclear shift, disappearance of eosinophils and eventual reduction of the number of lymphocytes and monocytes

(2) "The monocytic defense or subjection," in which there occurs a lessening in the number of leukocytes with decreased left shift, and an increase in the number of lymphocytes and monocytes with the reappearance of eosinophils

(3) "The lymphocytic cure," featuring the occurrence of lymphocytosis and eosinophilia and the subsidence of the nuclear shift

In acute infections with an unfavorable course there occurs only one phase; the second and third phases do not appear because regeneration does not take place The findings will probably be as follows: Increase in the number of immature neutrophils with increasing degenerative changes in the nuclei and cytoplasm; a decrease in the number of lymphocytes and monocytes with an absence or a decided decrease in the number of eosinophils.

In arranging a hemogram for determining the Schilling index, the most immature cells when present are listed first and the maturer types follow, so that the arrangement is from left to right—thus myelocytes, juveniles, stabs, neutrophils. A greater than normal percentage of immature cells constitutes a shift to the left.

Interpretation of the Schilling Nuclear Index: The total number of immature cells (*i e*, myelocytes plus juveniles, plus stabs) is divided by the total number of granular cells (*i e.*, myelocytes plus juveniles plus stabs, plus segmenters). Basophils and eosinophils are excluded.

Example: If differential count shows 70 per cent neutrophils of which five per cent are immature, Schilling index would show 70·5 = 0 07 or 7 per cent.

A degenerative shift, or a shift to the left, consists of a high increase of stabs and juveniles. It indicates a defective neutrophilic leukopoiesis such as is found in severe infections

A shift up to 15 per cent is normal, from 15 to 30 per cent denotes mild infection, a shift of 30 to 45 per cent indicates moderately severe infection, 45 to 60 per cent shift is to be found in severe infections and above 60 per cent shift to the left is an extremely grave prognostic omen

In a Schilling hemogram the following is to be noted:

(1) The total white cell count.

(2) The percentage of neutrophils.

(3) The morphology of the nucleus of the neutrophils.

(4) The percentage of basophils.

(5) The percentage of eosinophils
(6) The presence of unusual cells.
(7) Evidence of signs of degeneration in any of the cells.

The number of erythrocytes and the presence or absence of degenerative changes or of abnormal cells give additional information of the severity of the infection.

A Schilling hemogram is arranged as follows:

Count	B	E	M	J	St	S	L	Mon
20,000	0	0	4	26	30	20		

80 60 = 0 75 shift to left

B—basophils
E—eosinophils
M—myelocytes
J—juveniles
St—stabs
S—segmenters
L—lymphocytes
Mon—monocytes

luteum hormone, tuberculin, gelatin, peptone, bacterial toxins or heparin.

An *increase* in the number of platelets occurs in: Hodgkin's disease; chronic advanced tuberculosis; polycythemia, and *occasionally in Banti's disease.* The platelets usually increase in number after *splenectomy, blood transfusion, subcutaneous injections of blood,* of foreign protein or of some of the vitamins, and after strenuous exercise.

The Thrombocytes (Blood Platelets)

The blood platelets are said to be fragments of bone-marrow cells (*megakaryocytes*) *and are necessary constituents of the blood* Their average size is from two to four microns, some are larger They are well stained with Wright's or Giemsa's stains The platelet count in normal blood ranges from 150,000 to 500,000 to the cmm.; the average is about *300,000* to the cmm.

Function of the Blood Platelets: The *platelets and their products are concerned* with blood coagulation. A great diminution in their number will cause lengthening of bleeding and clot retraction time

The blood platelets are diminished in number (*thrombocytopenia*) in: Purpura; uremia; jaundice; anaphylactic shock; aplastic anemia; *Addison's disease*; measles; influenza; epidemic meningitis; *kala-azar:* and in malaria preceding the chill. Thrombocytopenia may occur as the result of the injections of calcium, benzol, tissue extract, corpus

Blood Grouping and Blood Typing

Human blood is grouped *into four* different types, according to the capacity *of their agglutinins to clump red* corpuscles The four types are variously named by Moss, Jansky and Landsteiner.

Systems of Nomenclature

Moss	Jansky	Landsteiner
IV	I	0
II	II	A
III	III	B
I	IV	AB

Since Moss' type IV corresponds to Jansky's type I, and Moss' type I corresponds to Jansky's type IV, therefore *when patient and donor have been typed* by different serologists, it is important to know whether the nomenclature employed by the two typers is the *same*

Landsteiner's classification O corresponds to Moss' IV, and Jansky's I because this type contains no agglutinin. A corresponds to both Moss' and Jansky's II because this type *contains*

agglutinogen A. B corresponds to Moss' and Jansky's type III and contains agglutinogen B. AB corresponds to Moss' I and Jansky's IV and contains agglutinogen A and B.

There are several sub groups of the main four groups and some bloods are Rh positive, others Rh negative. When these are mixed they hemolize. It is therefore desirable to match donor's and recipient's blood just before transfusion even though both belong to the same recognized blood groups.

Technic for Blood Matching

First Step: One cc of blood is obtained from a vein of the donor and of the recipient; one to three drops of blood of each specimen is placed in a separate test tube each containing one cc. of two per cent sodium citrate solution. The rest of the blood from each specimen is placed in individual dry test tubes which are allowed to stand or are centrifuged so as to obtain the serum

Second Step: A loopful of corpuscles from the patient's citrated blood is placed on a cover glass to which is added several loopfuls of the donor's serum from the noncitrated tube, and a loopful of the donor's corpuscles from the citrated tube is placed on another cover glass to which is added several loopfuls of the patient's serum from the noncitrated tube

Third Step: Each specimen is then examined under the microscope with a low power lens preferably as a hanging drop. If the two specimens of blood belong to the same group and match, no agglutination of red corpuscles will be noted in either specimen at the end of ten minutes.

Technic for Blood Grouping (Moss Classification)

The blood group to which an individual belongs is determined by testing his corpuscles and serum against the serum and corpuscles of an individual known to belong to blood group II or III. One to three drops of blood from the individual of the unknown group is placed in 1 cc. of two per cent sodium citrate in normal salt solution, and 1 cc of blood is placed in a dry test tube where the serum is separated from the corpuscles. The same procedure is carried out with the blood from a known group II individual.

A loopful of cells from the unknown group is placed on a slide and several loopfuls of serum from the group II is added, and a loopful of cells from the known group II is placed on another slide and a few loopfuls of serum from the unknown is added. Each slide properly covered is examined under a microscope with the low powered lens, and the agglutinations of the red cells are observed.

(1) If group II serum agglutinates the unknown corpuscles, and the unknown serum agglutinates the known group II corpuscles, then the unknown belongs to group III.

(2) If group II serum agglutinates the unknown corpuscles, and the unknown serum does not agglutinate the known group II corpuscles, then the unknown blood belongs to group I.

(3) If group II serum does not agglutinate the unknown corpuscles, and the unknown serum does agglutinate the known group II corpuscles, then the unknown belongs to group IV.

(4) If no agglutination occurs between either corpuscles or sera, then the

unknown blood belongs to the same group as the known, namely type II.

Behavior of Cells and Serum of Various Groups (Moss Classification)

Corpuscles of:

Group IV are not agglutinated by any serum.

Group II are agglutinated by serum of groups IV and III, but not by II and I.

Group III agglutinated by serum of groups IV and II, but not by III and I

Group I are agglutinated by serum of groups IV, II and III, not by group I.

Serum of:

Group IV agglutinates corpuscles of groups II, III, and I, but not group IV.

Group II agglutinates corpuscles of groups III and I, but not of groups IV and II.

Group III agglutinates corpuscles of groups II and I, but not IV and III.

Group I does not agglutinate any corpuscles

Therefore type IV Moss, type I Jansky and type O Landsteiner, is the universal donor and type I Moss, type IV Jansky and type AB Landsteiner, is the universal recipient.

Bloods of the same group and the same Rh that match are not agglutinable and are therefore chosen for transfusion. When the blood groups of the donor and recipient are not known, direct matching of the two bloods should be carried out.

The Rh Factor in Human Blood

The Rh factor was discovered in 1940 by Landsteiner and Wiener[1] while studying the cause of the severe hemolytic reactions occurring in rabbits after multiple transfusions with the blood of Rhesus monkeys. These authors observed that, after the first introduction of Rhesus red corpuscles into the bloodstream of rabbits, no reactions occurred, but that a subsequent transfusion produced a severe hemolytic reaction in the rabbit's blood. This reaction was found to be caused by a factor in the Rhesus monkey's red corpuscles which they named "Rh." The first transfusion produced antibodies in the rabbit's blood serum; and at the subsequent transfusions, the sensitized rabbit's serum reacting with the factor in the monkey's red cells hemolyzed the rabbit's blood. The rabbit's red cells are devoid of the Rh factor

The Rh factor is an antigen (allelomorph, allele, agglutinogen, or isoagglutinogen) present only in the erythrocytes and never in the plasma of certain persons It possesses the property of inducing the formation of antibodies (antiagglutinins) in the blood plasma of those whose red blood corpuscles are devoid of this factor

The Three Rh-Blood Types: The presence of three clinically important Rh factors has been identified in the red corpuscles of various persons, and the presence of several others, though of lesser clinical importance, is suspected. A person may have more than one type of Rh factor in his erythrocytes.

The three Rh factors are designated as Rh, also known as Rh^o or Rho, R'; and Rh''.

Rh^o is the most common, it is found in the red corpuscles of all the Rhesus monkeys (therefore termed by Landsteiner and Wiener "Rh factor"); in about 85 per cent of the white people, in 90 per cent of the negroes; in 90 to 100 per cent of the American Indians, Chinese, and Japanese; and in 93 per cent of the Asiatic Indians

[1] Landsteiner, K., and Wiener, A S . Proc Soc. Exper. Biol & Med, 43: 223, 1940.

Rh′ occurs less frequently. It is found in about 70 per cent of the white race, 20 per cent of the negroes; 85 to 95 per cent of the American Indians, Chinese, and Japanese; and 85 per cent of the Asiatic Indians

Rh″ is the least common. It occurs in about 30 per cent of the white people; 27 per cent of the negroes, 40 to 60 per cent of the American Indians, Chinese, and Japanese, and in 18 per cent of the Asiatic Indians

In England these types are known as C, D, and E

One, or two, or all three of these Rh factors may be present in the red cells of any one person. Eight distinct Rh-blood types are therefore possible: seven Rh-positives, and one Rh-negative. All have been shown to occur in human beings These are.

Rh°	Rh° Rh″	Rh′	Rh′ Rh″
Rh° Rh′	Rh° Rh′ Rh″	Rh″	Rh—

When more than a single Rh factor exists in the blood the combinations are more usually denominated as follows

Rh° Rh′	known also as Rh°′ or Rh_1
Rh° Rh″	known also as Rh°″ or Rh_2
Rh° Rh′ Rh″	known also as Rh°′″ or Rh_z
Rh′ Rh″	known also as Rh′″ or Rh_y

In human inheritance, the combinations Rh° Rh′ and Rh° Rh″ behave almost always as single units. This is the reason for the respective symbols "Rh_1" and "Rh_2"

The Rh-blood types, like the blood factors A_1, A_2, B, M, and N, have different distributions in different races Wiener,[2] in 1945, summarized the results of studies on the prevalence of the Rh-blood types among whites and negroes in New York City. He showed that, among the white population, it is rare for persons to have only one blood factor. Most Rh-positive whites have two or three factors in their red cells About 40 per cent of negroes, on the other hand, possess the single factor Rh° Almost no Rh-negative persons occur among the Chinese, American Indians, Filipinos, Hawaiians, and Australian aborigines. This is believed to explain why erythroblastosis fetalis is almost unknown in these races.

Persons possessing the Rh factor in their red blood cells are termed "Rh+" (Rh-positive). Those devoid of this factor are termed "Rh—" (Rh-negative) The Rh-negative patient when transfused with Rh-positive blood will develop antibodies in his blood (antiserum) which will agglutinate selectively the red cells of the various Rh-blood types Such sera are known as anti-Rh°, anti-Rh′, anti-Rh″, anti-Rh_1, anti-Rh_2, etc. Anti-Rh sera develop in Rh-negative patients who have been transfused with Rh-positive blood, or have received Rh-positive blood through other channels, such as is found in women who have recently given birth to infants with erythroblastosis.

It was found by Levine[3] and others that rarely will a serum of an Rh-positive person agglutinate all Rh-negative bloods and those Rh-positives which did not react with anti-Rh′ serum

Anti-Rh Sera: It was found that various maternal sera will agglutinate selectively the red cells of the different Rh-blood types Such sera are known as anti-Rh°, anti-Rh′, anti-Rh″, anti-Rh_1, anti-Rh_2, etc

The anti-Rh° serum is often referred to as "85-per cent" serum or "standard" serum, the anti-Rh′ as "70-per cent" serum, and the anti-Rh″ as "32-per cent"

[2] Wiener, A S Tr & Studies Phila Coll Phys, 13 105, 1945

[3] Levine, P, Katzin, E M, and Burnham, L J A M A, 116 825, 1941

serum. These percentages correspond to the Rh factors found in white persons.

Antisera have been prepared which will uncover the presence of several factors when *more than one is present*. The best known of these sera is the "87-per cent" serum, so-called because it contains antibodies against both Rh° and Rh′ and will agglutinate about 87 per cent of the red cells of white people. In practical experience, however, when testing unknown red cells for Rh composition, the simple anti-Rh°, anti-Rh′, and anti-Rh″ sera employed separately have proved more satisfactory than the more complex multiple sera.

The "8-per cent" anti-Rh° serum will pick out every one of the Rh-positive types except Rh′, Rh″, and Rh′ Rh″. These three types comprise less than 2 per cent of the white population, and less than 3 per cent of the colored. For this reason, and since specific anti-Rh′ and *anti-Rh″ serums occur infrequently and* often are of weak titer, the anti-Rh° serum is employed almost universally for routine screening of men and women for Rh-positivity. The anti-Rh′ and anti-Rh″ sera are used only when it is essential to determine the precise Rh genetic struc*ture, as in potentially incompatible hus*band-wife situations, in which erythroblastosis is feared in an expected infant, in anthropology, in medicolegal problems, and in a few other specific situations.

Agglutination and Blocking Antibodies: *There are two types of anti*bodies found in sensitized Rh-negative bloods.

1. The agglutinating antibodies or agglutinins will readily agglutinate Rh-positive red cells suspended in saline solution. These are unstable and may eventually disappear from the bloodstream These antibodies form in Rh-negative persons who were transfused with Rh-*positive blood.*

2 The blocking antibodies are more *stable They will* combine with the red cells, but will not clump them when suspended in saline; they will, however, agglutinate such red blood cells only when they are present in large numbers and are suspended in a viscous medium such as whole plasma or serum albumin. This type is more common in Rh-negative *women who have been repeatedly preg*nant with Rh-positive infants. When testing mother's blood for agglutinins, it is important to add the mother's serum so that blocking antibodies are not overlooked.

Hereditary Transmission of the Rh Factor: Two genes, one from the father and the other from the mother, determine the heredity of each separate Rh factor. It follows the mendelian law. When both *genes are positive, the patient is said* to be "homozygous," and is always Rh-positive. When one gene is positive and the other negative, the *individual is known* as "heterozygous," but he will still be Rh-positive, the positive gene being dominant and the negative recessive. The *various combinations of positive* and negative with the three separate Rh factors make possible twenty-one different Rh-positive constitutions. The genetic arrangement is determined by heredity, and remains constant throughout life.

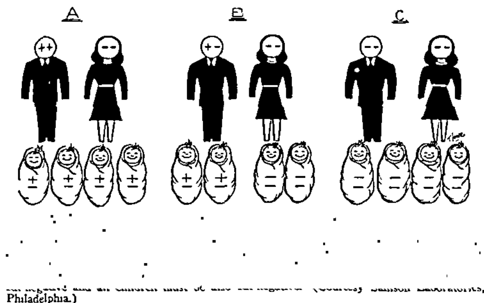
If the mother is Rh negative and the *father is homozygous* (Rh-positive), all their children will be heterozygous, though Rh-positive, since the positive *gene is dominant. When an* Rh-negative woman marries a heterozygous, though Rh-positive, man, half of their children *will be Rh-positive and the other* half Rh-negative. If both father and mother are

Rh-negative all their children will be Rh-negative.

Importance of the Rh Factor in Blood Transfusion: Transfusion reactions in Rh-negative persons who have received Rh-positive blood do not occur after the first transfusion and seldom after the second. These early transfusions sensitize the recipient's blood so that with each subsequent transfusion the hemolytic reactions become progressively more severe and may ultimately cause death. Rh-negative persons may be sensitized not only by intravenous transfusion but also when Rh-positive blood is given them intramuscularly, even though the quantity be small. Therefore Rh-negative persons should be transfused only with Rh-negative blood Levine and Waller[4] have shown that, once Rh-negative persons are immunized (sensitized) with Rh-positive blood, they remain potentially immunized for the rest of their lives.

Approximately 90 per cent of all hemolytic reactions following transfusions are caused, according to Unger,[5] by an incompatible Rh factor. He therefore advises that, even for Rh-positive patients, only Rh-positive blood should be used. Unger has further shown that if the patient lacks any one of the Rh factors, though he be Rh-positive, and the donor's blood contains the factor lacking in the recipient, the patient may become sensitized to this particular factor For transfusion purposes the blood of both the donor and the recipient should be investigated for their blood groups, i.e. O, A, B, and AB (see p. 1006), and for their Rh factors, so as to avoid incompatibilities. The donor's blood should also have a serologic test and other tests if infection is suspected.

It is of particular importance, when injecting blood intramuscularly or when transfusing women during their child-bearing period, young girls, or even baby



Philadelphia.)

[4] Levine, P , and Waller, R. K.: Blood, 1 143, 1946

[5] Unger, L. J : J Lab & Clin Med , 31, 793, 1946.

serum. These percentages *correspond to* the Rh factors found in white persons.

Antisera have been prepared which will uncover the presence of several factors when more than one is present. The best known of these sera is the "87-per cent" serum, so-called because it contains *antibodies against both Rh° and Rh′ and* will agglutinate about 87 per cent of the red cells of white people. In practical experience, however, when testing unknown red cells for Rh composition, the simple anti-Rh°, anti-Rh′, and anti-Rh″ sera employed separately have proved more satisfactory than *the more complex multiple* sera.

The "8-per cent" anti-Rh° serum will *pick out every one* of the Rh-positive types except Rh′, Rh″, and Rh′Rh″. These three types comprise less than 2 per cent of the white *population*, and less than 3 per cent of the colored. For this reason, and since specific anti-Rh′ and anti-Rh″ serums occur infrequently and often are of weak titer, the anti-Rh° serum is employed almost universally for routine screening of *men and women for Rh-positivity*. The anti-Rh′ and anti-Rh″ sera are used only when it is essential to determine the precise Rh genetic structure, as in potentially incompatible husband-wife situations, in which erythroblastosis is *feared in an expected infant*, in anthropology, in medicolegal problems, and in a few other specific situations.

Agglutination and Blocking Antibodies: There are two types of antibodies *found in sensitized Rh-negative* bloods.

1. The agglutinating antibodies or agglutinins will readily agglutinate Rh-positive red cells suspended in saline solution. *These are unstable and may eventually* disappear from the bloodstream. *These antibodies* form in Rh-negative persons *who were transfused with* Rh-positive blood.

2. The blocking antibodies are more stable They will combine with the red cells, but will not clump them when suspended in saline; they will, however, agglutinate *such red blood cells only when they are present in large numbers and are* suspended in a viscous medium such as whole plasma or serum albumin. This type is more common in Rh-negative women who have been repeatedly pregnant with Rh-positive infants. When testing *mother's blood for agglutinins, it is* important to add the mother's serum so that blocking antibodies are not overlooked.

Hereditary Transmission of the Rh Factor: Two genes, one from the father *and the other from the mother, determine* the heredity of each separate Rh factor. It follows the mendelian law. When both genes are positive, the patient is said to be "homozygous," and is always Rh-positive. When one gene is positive and *the other negative, the individual is known* as "heterozygous," but he will still be Rh-positive, the positive gene being dominant and the negative recessive. The various combinations of positive and negative with the three separate *Rh factors make possible twenty-one* different Rh-positive constitutions The genetic arrangement is determined by heredity, and remains constant throughout life.

If the mother is Rh negative and *the father is homozygous (Rh-positive)*, all their children will be *heterozygous*, though Rh-positive, since the positive gene is dominant. When an Rh-negative woman marries a heterozygous, though Rh-positive, *man, half of their* children will be Rh-positive and the other half Rh-negative. If both father and mother are

antigen or some agglutinins in the mother's blood other than an anti-Rh factor. Isoimmunization M, P, A, and O factors have been described.

The Hr Antigens: There are three Hr factors, namely, Hr°, Hr′, and Hr″. These bear reciprocal relationship to Rh°, Rh′, and Rh″. They can be identified by specific antisera, anti-Hr°, anti-Hr′, and anti-Hr″. The Hr factors and their antisera are important in the differentiation of a homozygous Rh-positive person from a heterozygous one In a *heterozygous person (mixed genes)*, his red cells will be clumped by both the anti-Rh and the anti-Hr sera for the specific factor. In a homozygous person, his red cells will be clumped only by anti-Rh serum and not by anti-Hr serum The Hr will take the place of the missing Rh The person who is Rh-negative is Hr-positive. The Hr indicates a factor opposite to the Rh because it is present in Rh-negative blood.

Parental Precautions: Soon after a woman becomes pregnant, her blood should be tested for its Rh factor. If positive, further tests are usually unnecessary. If she is an Rh-negative, the husband should also be tested If he, too, is Rh-negative, further tests are unnecessary But if the husband is Rh-positive and the wife is Rh-negative, the wife's blood should be tested for antibodies during her fifth month of pregnancy, and repeated during the seventh, eighth, and again in the ninth month. A rising titer is a danger signal that the baby may be erythroblastic If the mother's blood shows a rising titer, the father's red cells are to be examined to determine whether he is homozygous or heterozygous If the father is heterozygous, the baby has an even chance of escaping erythroblastosis as it may be Rh-negative But if the father is homozygous, the baby is bound to be Rh-positive and may be born with erythroblastosis. Soon after birth, every baby born of an Rh-negative mother and an Rh-positive father should have its Rh factor determined. If it is Rh-negative, erythroblastosis need not be feared But if it is Rh-positive and shows signs of erythroblastosis, transfusion of citrated blood, preferably Rh-negative blood or washed Rh-negative red corpuscles should be given as often as necessary. The quantity is usually ½ oz. per *pound or 30 cc. per kg*

Breast feeding of an Rh-positive infant by an Rh-negative mother, particularly if her blood contains antibodies, is a dangerous procedure.

Transfusion Reactions

Transfusion reactions may be due to nonspecific causes or to specific incompatibilities.

Nonspecific Transfusion Reactions: Mild transfusion reactions, such as headache, mild chill, and slight rise in temperature may occur after transfusion with properly tested compatible blood, or after any intravenous injection. However, repeated transfusions from the same donor, though the recipient's and donor's blood are properly matched for routine transfusion, may, after the third or fourth transfusion, cause a hemolytic reaction because of the development of isohemolysins in the recipient's blood Allergic reactions, such as headache, itching, urticaria, and edema may be caused by the sensitivity of the recipient to the donor's blood protein, or the recipient's sensitivity to something the donor had eaten prior to the transfusion Transfusion at a too rapid rate may cause a sense of fulness in the peripheral circulation, coughing, fulness in the head, and vertigo

girls, that the blood given them should be Rh-compatible so as to avoid lifelong sensitization and prevent the possibility of later bearing erythroblastic children.

The Effect of Blood Plasma Injections: While it is of utmost importance, when administering whole blood, to be meticulously careful to select compatible Rh factors as well as A and B groups, such a selection is not necessary when blood plasma is given. The Rh factors *reside only in the red cells; therefore* plasma, irrespective of its source, may be transfused alike in those classified as Rh-positive or Rh-negative.

The Role of the Rh Factors in *Pregnancy:* *An Rh-positive husband* may sensitize an Rh-negative pregnant wife when the fetal blood enters her circulation. Because an Rh-positive father transmits a positive gene to his offspring, the fetus becomes Rh-positive, so that when this fetal blood enters the mother's *circulation, which is Rh-negative, it* causes the formation of antibodies in the mother's serum The small quantity of fetal blood entering the mother's circulation may sensitize her blood and form an antiserum which is not sufficient to give her a hemolytic reaction, but her anti*serum, on entering the fetal circulation* through the placenta, may attack the red cells of the fetus and cause agglutination and hemolysis which may result in erythroblastosis and allied conditions

When transfusion becomes necessary during pregnancy or soon thereafter, both the mother's and father's blood should be carefully tested for their Rh types as well as *her blood group, and only matching* blood should be used.

Effect of Dissimilar Parental Rh Factors Upon the Offspring: Infants born with erythroblastosis, congenital hydrops, icterus gravis, kernicterus, prematurity, splenomegaly, idiopathic anemia, and some forms of feeblemindedness may be the result of the mating of an Rh-positive father with an Rh-negative *mother who had previously been sensi*tized. If the mother has not previously been sensitized, the baby will be normal.

Erythroblastosis fetalis and its variants do not occur with the first pregnancy and *seldom with the second, unless the mother* had previously been sensitized by the transfusion of unmatched blood which caused her to develop agglutinins in her serum. The subsequent passage of these maternal specific antibodies through the placenta, and their continuous action on *the susceptible fetus, will cause hemoly*sis of the fetal blood which is the characteristic feature of this disease

The majority of cases of erythroblastosis occur with the third, fourth, or later pregnancies. The previous pregnancies, like the first transfusion, may sensitize *the mother's blood, but not* be sufficient to cause hemolytic reactions in the fetus. *It is only after a sufficient number of anti*bodies have formed in the maternal blood that it becomes capable of affecting the fetal circulation. Over 95 per cent of the Rh-negative mothers *do not* give birth to erythroblastic children.

It is estimated that only 2 to 4 per cent of *Rh-negative mothers who have* Rh-positive husbands give birth to erythroblastic babies. It was shown *by Polayes* and Ohlbaum[6] that erythroblastosis fetalis is not always the product of an Rh-negative mother and an Rh-positive father, since about 10 per cent of mothers of erythroblastic babies in their series were Rh-positive. However, the fetus *and the mother were of* different Rh types. The cause of this may be an Hr

[6] Polayes, S H, and Ohlbaum, C.: Am. J. Clin. Path, 15. 467, 1946.

Collecting of Specimens for Laboratory Diagnosis

Determination	*Type of Specimen*	*Stability Hours*	*Normal Average*	*Value Range*
Arsenic, heavy metals	Qualitative· 25 cc urine,		Quantitative	24-hr specimen
		1	1 mg /100	0 4–2 5
		24	4 8%	3 8–5 5
		3	60	25–125
			Less than 1 mg / 100 cc	
Barbiturate, urine	25 cc , or more urine		Best for detection of drug	
Bilirubin, serum	10 cc clotted blood	6	0 3 mg /100	0 2–0 5
Bromide, serum	10 cc clotted blood	6	Trace	
Bromsulfalein	5 cc clotted blood, 30 minutes after 5 mg /kg	24	3%	0–10%
Calcium, serum	10 cc clotted blood	6	10 mg /100	9–11 5
Calcium, ionized	10 cc clotted blood	6	4 8 mg /100	4 3–5 3
Cephalin, flocculation	5 cc. clotted, unhemolysed		0 to +	
		2	50–60 vol %	22–29mEq
		4	570–620 mg /100	97–106mEq
			0–0 2%	NaCl urine
			A=over 30	
Volume Test	morning urines, 1 oxalated, 1 clotted blood			
Cholesterol, serum	5 cc clotted blood	24	210 mg /100	100–350
Cholesterol, ester	Same specimen, if requested	6	50–80% of total	
Congo red test	10 cc clotted blood, 4 and 60 minutes	24	10–40% disappearance	
Creatinine, blood	5 cc oxalated blood		1 0–2 0 mg /100	
Globulin, serum			2 4%	1 5–3 4
Fibrinogen, plasma	10 cc oxalated blood	24	0 2–0 4%	
Glucose tolerance, 2 dose	3 cc oxalated blood, fasting ½, 1 hr		No specimen over 160	
Glucose, blood, new method	3 cc oxalated blood, fasting	1	105 mg /100	85–125
Glucose, blood	3 cc oxalated blood fasting	1	90 mg /100	70–110
Hemoglobin	1 cc oxalated blood, no clots	24	Males 15 6, females 14 5	
Hippuric acid test	4-hr specimen urine	6	3 5 gm	2 5–4 5
Icterus index	5 cc clotted, unhemolysed blood	24	6	4–8
Lead, whole blood	6 to 8 cc clotted blood special tube		0 03 mg /100	0 01–0 06
Lipase, serum	5 cc clotted blood	6	0 3–1 3 cc	N/20 NaOH
Magnesium, serum	10 cc clotted, unhemolysed blood	6	2 0 mg /100	1 0–3 0
Non-protein N	3 cc oxalated blood	24	30 mg /100	15–40
pH, plasma	10 cc oxalated under oil, chill		7 38	7 33–7 45
Phosphate, inorganic	5 cc. clotted, fasting blood	4	Adult 3–4 5, child 5–6	
Phosphatase, serum, alk	10 cc clotted, fasting blood	4	Adult 1 5–4, child 3–13	
Phosphatase, serum, acid	10 cc clotted, fasting blood	4	0–1 1 (Shinowara-Jones Reinhart)	
Protein, serum, total	5 cc clotted, unhemolysed blood	24	7 2%	6 3–8 0
Protein, urine, quantitative	24-hr specimen toluene (urine)		0 to 0 1 gm per 24 hrs	
Potassium, serum	7 cc clotted, unhemolysed blood	4	19 mg /100	18–22
Prothrombin, plasma	4 5 cc prothrombin tube	2	100%	70–100+
Sodium, serum	6 cc clotted blood	4	140 mEq/1	128–151
Specific gravity, serum	5 cc clotted blood		1 027	1 025–29
Spinal fluid chloride	2 cc spinal fluid	24+	700–750/100	120–128mEq
Spinal fluid protein	3 cc , no blood, spinal fluid	24	30 mg /100	15–45
Spinal fluid sugar	2 cc spinal fluid	4	60 mg /100	50–75
Sulfanilamide, etc	5 cc oxalated blood	24+	0	
Takata-Ara	5 cc clotted blood	6	Negative	
Thiocyanate	8 cc clotted blood	24+	Trace	
Total base	5 cc clotted blood	4	160mEq/1	155–70
Urea N, blood	3 cc oxalated blood	24	10–16	6–22 mg /100
Urea clearance	3 cc blood, timed urine	24	Over 70%	
Uric acid, serum	10 cc clotted blood	6	Males 2 5–5 5, female 1 3–4	
Van den Bergh, qualitative	5 cc clotted, unhemolysed blood	6	Negative or weakly positive	
Wassermann, blood	5 cc clotted blood	24+	Negative	
Wassermann, spinal fluid	2 cc spinal fluid			
Colloidal gold	spinal fluid			

When this occurs, the transfusion is to be stopped, and may be continued later at a very slow rate, such as 40 drops to the minute. These reactions are non-specific; they do not cause unusual discomfort and are seldom fatal.

Specific Transfusion Reactions (Hemolysis): These reactions are severe and may be fatal. They are caused by the introduction of incompatibilities in the bloodstream These incompatibilities may be the following:

1. A different blood group than that to which the patient belongs.
2. A difference between the Rh factors of the donor and recipient.
3. An Rh-positive blood in a sensitized Rh-negative individual.
4. The use of stale blood (blood from a bank older than five days).
5. The presence of pyrogens and other hemolysins in the blood.

Symptoms of Specific Reaction (Hemolysis): Symptoms begin to appear soon after a small amount of blood, usually about 50 cc., or less, has been introduced. The patient becomes anxious and will complain of a tingling sensation, general discomfort, fulness in the head, precordial oppression and pain, difficulty in getting his breath, and severe pain in the back of the neck and in the lumbar region.

Physical Signs: The pulse becomes slow at first and then rapid and thready. The skin is cold and clammy, and there is marked flushing of the face and general cyanosis. The temperature may rise to 105° F. (41° C.) which is preceded by a severe chill. Dyspnea becomes marked The blood pressure falls. The pupils become dilated, and soon nausea and vomiting set in. Within a few hours, there may be hematuria or entire suppression of urine, and later jaundice may develop. The development of jaundice and of anuria is a danger signal, since death may follow. Death may be due to the formation of multiple thrombi in the blood vessels, as well as in the capillaries, to exsanguination resulting from hemorrhages caused by the hemolytic reaction, or to vasoconstriction, the so-called "reflex anuria." In the case of prolonged anuria, Neurwit advocates splanchnic block; others advocate spinal anesthesia.

Blood Chemistry

Blood chemistry is employed for metabolic investigation, for diagnosis, differential diagnosis, prognosis and treatment of disease. Many of these tests can be carried out by technicians provided they are supervised by a physician with the requisite laboratory training or by a practical biochemist preferably with hospital or clinical experience.

Time for Collecting Blood: Every physician is familiar with the details of collecting blood. There is one point to be emphasized, namely, the necessity of using a very sharp needle. The majority of new needles are quite blunt and it is advantageous to sharpen them The necessity for thorough surgical asepsis, and the locating of a suitable vein are too well known to merit description here.

The best time for collecting blood is in the morning before breakfast. A few crystals of potassium oxalate[1] will prevent coagulation and a pinch of potassium

[1] Obviously potassium oxalate cannot be used if the calcium or potassium of the blood is to be determined (oxalate precipitates the calcium in the blood which is necessary for coagulation). In such cases sodium citrate is used.

Composition of Normal Blood and of the Blood in Certain Pathological Conditions[1]

(After *Hawk.*)

	Normal	Chronic Nephritis	Uremia	Early Diabetes	Severe Diabetes	Moderate Acidosis	Severe Acidosis	Gout	Lipemia	Chole-lithiasis	Arthritis
Total solids, per cent.	20 0	13–19	12–18		17–20		...	19–21	...	...	...
Total N per cent.	3 0	2 5–3 0	1 7–2.7		1.8–2 9	...	...	...	...	...	...
Nonprotein N.	25.30 or 35	30 90	90–400	...			...	25–35	...	...	60–100
Urea N.	12–15	16–70	70–300	...	...	...	...	...	...	...	...
Uric acid	2–3 5	3–10	4–25		...	...	...	4–10	...	...	2–8
Creatinine	1–2	2–4	4–35				...	...	...	...	...
-Creatine	3–7	...	7–30			...	...	...	...	...	...
Amino-acid N.	6–8		8–30	...		...	...			...	...
Ammonia N.	0 1–0 2	0 1–0.2	0 2–1 0					...	...	...	
Sugar, per cent.	0 08–0 12	...	0 1–0.2	0.14–0 30	0 3–1 2	...	...	...	...	...	...
Acetone plus acetoacetic acid	0–1 0	...	2–25	1.5–12	10–40	...	...	...	...		...
B-hydroxybutyric acid	0–3 0	...	5–25	5–15	10–100			...	...	...	...
Alkali reserve (c.c. CO_2 in 100 cc. plasma)	77–53					40–30	Below 30	...	...	...	...
Cholesterol	140–180	170–350	170–350		150–300	...	...	...	500–3600	280–950	...
-Chlorides as NaCl per cent	0 65	0 55–0 75	0 45–0 65	...	0 60		...	...		...	...
Acid soluble phosphorus	2 6	3–7	7–21	...		...	...	...		...	...
Lipoid phosphorus	6–12	8–13	8–30	...		...	...	...	...	...	...
Fat, per cent.	0 1–0 7[2]	...			3–18	...	...	...	3–29	...	
Calcium (plasma)	10	...	3–9	...	...	...	...	...	...	...	...

[1] Results are expressed as milligrams per 100 cc of blood, unless otherwise indicated. Some of the figures given are based upon but few analyses and may not be entirely characteristic

[2] A short time after a meal rich in fat, the blood may contain considerably more fat.

fluoride may be added as a preservative to the five or ten cubic centimeters of blood.

Physical Examination of the Blood: Valuable information can be obtained by the inspection of freshly drawn samples *of blood*, as for instance, its viscosity, *and the rapidity of erythrocyte sedimentation.* (SEE: Blood Sedimentation Test *on page 997*.) *The relative* volume of blood plasma should be observed in anemias, especially before making a diagnosis of polycythemia. In leukemias the total white count can be roughly estimated by the thickness of the sedimented film of white corpuscles. It is *important to look for abnormal pigmentation* of the plasma such as bile pigments. *Most essential is the observation* of the color of the venous blood itself as follows herewith.

In all cases of uncomplicated acidosis and in any condition where the bases in the blood are insufficient to carry the normal amount of carbon dioxide, the venous blood becomes arterialized in color. The extreme arterial color of venous blood can be visualized in the terminal picture of pure diabetic acidosis *It is well known that blood becomes* darker in color as the oxygen is replaced by carbon dioxide, and venous blood resembling arterial ought not to be overlooked as a possibility of hypooxidation. In short, there is such a thing as physical examination of blood which should not be neglected

With the above mentioned advantages of making gross observation of blood and plasma in mind, blood chemistry may now be considered See Table of Blood Chemistry Values in Health and *Certain Diseases, p. 992.*

An inspection of the table on p. 1009 shows *the range of values in chronic* nephritis, uremia, early and severe diabetes, moderate and severe acidosis, gout, lipemia, cholelithiasis and arthritis

In addition blood chemistry studies should be obtained in all preoperative bladder and *prostate conditions.* The diagnosis as well as the prognosis of *toxemias of pregnancy can be better followed* by determining the blood chemistry at midterm. In pneumonia cyanosis is now correlated with venous and arterial unsaturation. In major fractures when union does take place there is a rise in the inorganic phosphorus content of the blood, but in nonunion there is no rise in blood phosphorus.

The *use of blood sugar estimations* to control the insulin dosage is well *known, and likewise, the determination* of plasma chlorides as a guide to the diagnosis and treatment of hypertension

Pathologic Changes in the Blood Chemistry

Under normal conditions the various chemical substances found in the blood occur in the proportions given in the table The nitrogenous bodies, such as urea, nonprotein-nitrogen and uric acid *are usually found in increased* amounts when the kidneys fail to excrete them, and therefore their increase in the blood indicates the degree of kidney dysfunction. *Both in acute and chronic interstitial* nephritis these products are invariably retained

Nonprotein Nitrogen: This includes the nitrogen present in urea, uric acid, creatinin, ammonia and, in fact, all nitrogen in a nonprotein form. Normal, whole blood contains 25 to 35 mg. of nonprotein nitrogen to the 100 cc. An increase over this amount is an indication of kidney inefficiency. A gradual increase of this substance in the blood on a low

diminution of either the albumin or the *globulin, or an entire reversal* of the albumin-globulin ratio.

Hyperproteinemia: An increase of albumin alone in the blood plasma occurs in but few conditions. The general increase of protein is due to an increase of the globulin fraction. In some of the acute and chronic infections and suppurations, the total blood protein is increased, the *globulin often being two or three times as high as the albumin;* this is noted in pneumococcic pneumonia, rheumatic fever, rheumatoid arthritis, subacute bacterial endocarditis, leprosy, kala-azar, Boeck's sarcoid, multiple myelomata, myelogenous leukemia, osteomyelitis, lung abscess, lymphogranuloma, in various chronic suppurative diseases, in filariasis, trypanosomiasis, Schistosomiasis, and at times in malaria, tuberculo*sis and syphilis. Hyperproteinemia oc*curs also in acute dehydration, and may be found in severe vomiting, severe diarrhea, cholera, extensive burns, Addison's disease, intestinal fistula. According to H. A. Reimann, prolonged high globulin content of the blood plasma associated with chronic suppurative processes is often responsible for amyloid disease.

Hypoproteinemia: The decrease of plasma protein occurs chiefly in the albumin fraction; this may be accompanied by a relative increase of the globulin fraction as a compensatory measure for the primary deficiency.

A decrease of serum protein is a constant and significant finding in *all* types of edema This, according to Trumper and Cantarow, is the result of a diminished plasma colloid osmotic pressure within the blood vessels which decreases the ability of the plasma to hold water and causes an extravasation of water into the tissues. Depletion of plasma proteins from any cause results in edema. The degree of edema and the time of its appearance depends more upon the concentration of the albumin fraction than upon that of the globulin fraction.

Diminution of total plasma protein with a decrease of the plasma albumin and a compensatory increase of globulin that is a reversal of the albumin globulin ratio and a decrease in the fibrinogen *occurs in the following conditions* Chronic nephritis with marked albuminuria; portal cirrhosis, hepatocellular diseases; inanition, and lipoid nephrosis. It also occurs in toxemia of pregnancy, in primary disturbance of protein metabolism and where regeneration of serum albumin is interfered with.

Prothrombin: SEE: p. 912.

Fibrinogen: The normal fibrinogen content of plasma is 0 2 to 0.4 mg. to 100 cc.

The fibrinogen content of the plasma is *increased* in nephrosis, in most of the acute fevers (except typhoid), *i. e*, lobar pneumonia, septicemia, bacteremia; in infections, such as sinusitis, tonsillitis, acute appendicitis, cholecystitis; in multiple myeloma, lymphogranuloma inguinale and in certain diseases of the liver *It is also increased during pregnancy* and menstruation, and following x-ray treatments

Decreased plasma fibrinogen occurs in typhoid fever, acute hepatic insufficiency, such as caused by chloroform, arsenic, phosphorus and tetrachloride poisoning, and in acute yellow atrophy of the liver. It is also decreased temporarily after severe hemorrhage and occasionally in malignancy.

Chlorides: Normal whole blood contains 400 to 500 mg. of chlorides to the 100 cc. The normal blood plasma contains 570 to 620 mg. to the 100 cc.

nitrogenous diet indicates a progression of the lesion, especially if the creatinine also increases. The graver the lesion the greater is the retention of this substance in the blood. In uremia 400 mg. of non-protein nitrogen, or more, may be found in the blood.

Blood Urea Nitrogen: Urea is the chief end product of protein metabolism; it is freely excreted by the kidneys. The total urine urea depends upon the amount of protein ingested; the higher the protein intake the greater is the quantity of urea eliminated in the urine. On an average diet, about 30 Gm. of urea is eliminated in 24 hours which is 50 per cent of the total urinary solids. Normal blood contains 12 to 15 mg of urea nitrogen to 100 cc. A quantity above 15 mg. in 100 cc. of blood indicates retention. *In glomerular nephritis, the urea* may mount up to 30 or 60 mg. or more. In uremia it may be as high as 175 or 300 mg. to 100 cc. of blood. In normal *blood 50 per cent of nonprotein nitrogen* is in the form of urea. In uremia the percentage is increased, and the other bodies, such as uric acid and creatinine, are *also increased but not proportionately.*

An *increase* of urea nitrogen in the blood is found in severe kidney damage, in urinary retention due to disease of one or both kidneys, prostatic obstruction, or other condition that will interfere with urinary excretion. It is also increased in acute intestinal obstruction, excessive vomiting, severe dehydration and hemoconcentration and in severe liver damage, in advanced stages of osteitis fibrosa cystica, and occasionally in diabetic coma.

A *decrease* of urea nitrogen in the blood may be found during the sixth, seventh, and eighth months of normal pregnancy, also in nephrosis, acute hepatic insufficiency due to chloroform, phosphorus or arsphenamine poisoning, and in acute yellow atrophy.

Uric Acid: This substance is poorly soluble, therefore an increase in the blood above the normal may occur in early nephritis, before N.P.N. urea and creatinine are retained. *Normal blood* contains from *2 to 3.5 mg. of uric acid* to 100 cc. In nephritis the quantity may be increased to 10, 20 or 30 mg. to 100 cc. of blood. Gout, and some forms of *arthritis, even in the absence of a kidney* lesion, may show from 5 to 10 mg. of uric acid to the 100 cc. of blood. Other conditions in which there is an increase of uric acid in the blood are leukemia, multiple myeloma, lead poisoning, intestinal obstruction, impaired hepatic function, osteoarthritis, cardiac decompensation, and pregnancy.

Creatinine: Creatinine is an anhydrid of creatine which is normally found in muscle. It is a product of endogenous *protein metabolism and its quantity* in the blood is little affected by diet. This substance is more freely excreted by the kidneys than any other form of nitrogen; *therefore*, a *retention* of creatinine in the blood is an indication of grave kidney insufficiency. Normal blood contains 1 to 2 mg. of creatinine to 100 cc. of blood. Above 4 mg. of creatinine to the 100 cc. of blood indicates kidney impairment. In uremia creatinine concentration may be increased to 10, 18 or more mg.

Plasma Proteins: The normal plasma protein is from 6.5 to 8.5 Gm. per 100 cc. *This is made up chiefly of* albumin 4.6 to 6.7 and globulin 1.5 to 2.5. The albumin-globulin ratio is maintained in health. In disease the plasma protein as a whole may be increased, or diminished, or there may be an increase, a

Very often, *preceding diabetic coma,* a marked hyperglycemia exists, which in itself is not so serious. But if there is also acidosis, that is, a marked increase in acetone bodies from incomplete fat metabolism (with dyspnea and without cyanosis) resulting from the excessive withdrawal of bases from the blood, it indicates grave danger This diagnosis is best confirmed by an estimation of either the carbon-dioxide combining power of the blood plasma, the hydrogen-ion concentration or pH of the blood plasma or, if the patient is able to cooperate, the carbon-dioxide content or tension of the alveolar air may be ascertained.

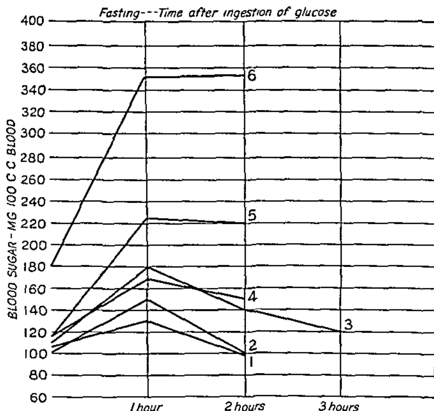


Fig 2—Glucose tolerance chart

Interpretation of Table

1
2. '
3
4
5 Mild diabetes
6 Low sugar tolerance, indicative of advanced diabetes

Acidosis and Ketosis

Ketone Bodies (acetone bodies): An excessive accumulation of these bodies in the blood will cause acidosis or ketosis By acidosis is meant a condition brought about by the excessive withdrawal of al-

The chlorides in the blood may be especially *increased* in nephritis with edema, diabetes, anemia, certain fevers and at times in lobar pneumonia and in a large per cent of cases of hypertension. The chlorides are *diminished* in severe vomiting, pyloric obstruction, achlorhydria, uremia, nephritic acidosis, edema, emphysema, adrenal cortical insufficiency as in Addison's disease, following operative procedures, particularly upon the gastrointestinal tract, and in those subjected to high temperatures who sweat profusely and drink large quantities of salt-free water.

Potassium: Normal blood contains 150 to 250 mg. to 100 cc. of whole blood and 16 to 22 mg. to 100 cc. of plasma. The potassium is *increased* in uremia, in eclampsia and in Addison's disease at the expense of the chlorides. Potassium depresses the function of the myocardium, dilates the coronaries, stimulates the vagus, and may cause tetany by producing alkalosis

Glucose: Normal blood contains from 80 to 100 mg. of glucose to 100 cc. of blood. An increase of sugar in the blood (hyperglycemia) is found in diabetes mellitus, in mild cases, 140 to 300 mg ; in severe cases as high as 400 to 600 mg to 100 cc. of blood Mild hyperglycemia may be found in Addison's disease, hyperthyroidism, pancreatic disease and in disease of the other endocrine glands. Normally, sugar begins to appear in the urine when the blood sugar concentration reaches 150 to 180 mg to 100 cc., which concentration is considered as the normal renal threshold.

Sugar Tolerance Test: After a fast of at least 12 hours, about 5 cc. of blood is drawn from a vein and its glucose content is determined. (This is best done in the morning after an all-night's fast.) Then the patient is given a solution of dextrose containing 0.8 Gm per pound of body weight. The glucose should be dissolved in 500 cc. of water flavored with lemon.

At the end of one hour another specimen (second specimen) of blood is drawn and examined, and an hour after that, a third specimen is taken

Interpretation Normally, the fasting blood sugar is 112 mg or less One hour after the ingestion of the proper amount of glucose, the blood sugar reaches a height of 150 to 160 mg. per 100 cc., but at the end of the second hour it returns to the fasting level.

In *diabetes mellitus*, the fasting blood sugar may be within normal limits or above, depending upon the severity of the case, but at the end of the first hour after the glucose ingestion, it reaches a height much above the kidney threshold for sugar (170), and its return to the fasting level is slow, so that, at the end of the second or even the third hour, the blood sugar is still of a high enough concentration to cause glycosuria

In *hyperfunction of the adrenals, pituitary or thyroid*, the fasting blood sugar is normal or slightly elevated, and, after the glucose ingestion, it rises only slightly above the kidney sugar threshold, and drops considerably within the first hour, but does not return to the fasting level until three or four hours later.

In *hypofunction* of the above mentioned glands, the fasting blood sugar is normal, rises only slightly above the highest normal level (120 mg. per 100 cc. of blood) and returns to the fasting level within the second hour after the glucose ingestion.

In *renal glycosuria*, the blood sugar is always within normal limits or below, in spite of a constant mild glycosuria.

I. Hydrogen-ion concentration of the blood plasma. *II. Alkali reserve of the* blood plasma (Van Slyke). III. Alkali tolerance of the patient. IV. Carbon-dioxide tension of the alveolar air.

I. ***Hydrogen-ion Concentration:*** In making H-ion determinations, electrometric and colorimetric methods are available. Since the electrometric method requires elaborate equipment and an operator with considerable training, the discussion will be confined to the colorimetric method, which is commonly used clinically.

Each indicator that is used has its own definite *p*H range. For example, bromthymol blue covers the range *p*H 6 0 to 7 6. If the *p*H value of a solution to which bromthymol blue is added is 6 0 or below, the indicator will be yellow If the *p*H of the solution is 7 6 or above, the indicator will be deep blue Between these two points the color will range from yellow to blue, depending on the *p*H of the solution.

Buffers: If acid or alkali be added to a solution of a strong base or acid, it will be found that usually the *p*H is markedly affected. Certain substances, however, when present in the solution, act to modify this usual effect in such manner that the changes in *p*H may be practically inappreciable Such substances are known as buffers, and they are quite common in biological fluids These properties of buffered solutions are made use of in the colorimetric method for determining hydrogen-ion concentration By mixing certain solutions in definite proportions, mixtures are prepared of definite *p*H values A suitable indicator is then chosen and added to these mixtures and to the unknown A rough estimate of the *p*H of the unknown can be obtained by systematically testing it with different indicators, for by this it is shown exactly at what *p*H the maximum acid or alkaline color may be expected The exact proportions in which these buffer salts must be mixed to obtain desired *p*H values can be found in all standard manuals

The *colorimetric method* of Cullen is widely used clinically with some modifications. The *electrometric method* of determining the hydrogen-ion concentration is primarily used for investigation and seldom for clinical purposes unless as a check against the colorimetric method.

The pH *of the Blood is Remarkably Constant:* For example, an arterial blood of which the *p*H = 7 35 (average normal) may change as it becomes venous blood to *p*H = 7 34 or possibly = 7 32 The lowest *p*H yet reported in man with recovery from acidosis was a *p*H of 6 98 and 7 02 In a case of nephritic acidosis with a *p*H = 6.7 with strenuous alkali therapy for 36 hours, the acid balance returned to a *p*H of 7 25 and the patient lived 48 hours The range compatible with life probably lies between *p*H of 7 0 and 7.8

The average value for normal urine is *p*H6, while for gastric juice, which is the most acid secretion in the body, the *p*H is 1 7

II ***Alkali Reserve of the Blood:*** The alkali reserve of the blood bears a definite relation to that of the entire body The average value for man is 65 volume per cent of carbon dioxide. According to Hawk and Bergeim[1] the normal adult's reserve of bicarbonate is 80 to 53 volume per cent, in mild acidosis, with no previous symptoms, 53 to 40 per cent; in moderate to severe acidosis,

[1] Hawk and Bergeim · Physiological Chemistry, 8th Edit.

kalies through the formation of fixed acids which can only be eliminated by the kidneys, or by the retention of acids within the body. Recognition of acidosis plays an important part in such diseases as diabetes mellitus, severe nephritis, food intoxication and diarrhea with vomiting, and in hyperemesis gravidarum.

Normally, the body is in a state of compensated acidosis and is protected against acidosis in various ways according to the following summarization by Dr. Campbell of Toronto:

1. By the proper balance of available carbohydrate in the food against the protein and fat. (Antiketogenic *vs.* ketogenic)
2. Selecting foodstuffs not too high in protein because the proteins, when burned, yield phosphoric and sulfuric acids.
3. Selecting foods containing an excess of inorganic bases over inorganic and organic acids.
4. By an adequate supply of fluids (ketonic acids may be excreted in dilute form).
5 By production of ammonia, which neutralizes acids and conserves sodium, the essential base to carry CO_2.
6. By the combination of the calcium and magnesium of bone with acids (neutralization).
7. Excretion of buffer salts; bicarbonates and phosphates.
8. Abnormally rapid excretion of carbon dioxide from the lungs.
9. By the use of proteins in the blood or tissues as acids or as bases for combination, to avoid change in reaction.

If one of the above mechanisms is not used during the course of a disease, the others may rectify ensuing errors in metabolism, but in severe diabetes a number of these mechanisms are ineffective.

As long as the acid-base equilibrium or *p*H is normal, there is compensated acidosis. Van Slyke restricts the use of the term acidosis to describe the condition caused by acid retention sufficient to lower either the bicarbonate or the *p*H of the blood below normal limits The subject of acidosis is too recondite to be thoroughly presented here, but a few basic clinical observations will be mentioned. It is well to remember Yandell Henderson's simple test: A normal person can hold the breath from 30 to 40 seconds without an especially deep inspiration, but this period diminishes in proportion to the reduction of the bases in the blood. In acidosis, the blood tests are more dependable than the alkali tolerance or the alveolar carbon-dioxide tension tests, because in the latter it is not possible to obtain the coöperation of the semi- or completely comatose patient. It is well to remember that in profound diabetic coma, the high renal threshold which is often present (the higher this threshold, the more serious is the prognosis), prevents little if any sugar spilling over into the urine, even in instances where the blood sugar concentration is around 300 milligrams, whereas in uremic coma it is common to find a trace of sugar in the urine. This makes it difficult to differentiate between diabetic coma and uremic coma unless blood tests are made.

Test for Diagnosis of Acidosis: Ordinarily, the diagnosis of acidosis can be made or confirmed by any one of the following tests (if the patient is verging on coma, it is necessary to make one of the blood tests that does not require this coöperation):

based upon the absorption, by means of potassium hydroxide, of the carbon dioxide from a known amount of alveolar air. The average normal value for men is 55 to 65 volumes per cent. In women and children the normal value is lower. In the presence of acidosis the amount of carbon dioxide falls, and may be as low as 20 per cent or lower. In cases of diabetic coma, below 20 per cent is a danger signal of the oncoming of acidosis. The details of this test can be found in all laboratory manuals.

Ketosis is a form of acidosis due to overproduction of acids of the *ketone group*, *e g*, betaoxybutyric and aceto-acetic acids The ketone acids in the body are the end product of fat metabolism requiring one molecule of sugar to two molecules of these acids to be finally broken up into carbon dioxide and water

The most striking clinical sign of acidosis is hyperpnea (very deep regular and continuous breathing)

Alkalosis

By alkalosis is meant a condition in which there is an increase in the alkalinity of the blood. This condition may be brought about by either an excessive accumulation of alkalies in the blood or an excessive withdrawal of acids or chlorides from the blood The ion concentration or the reaction of the blood depends upon the ratio of H_2CO_3—$NaHCO_3$, therefore an increase in the bicarbonates will lead to alkalosis and an increase in carbonic acid to acidosis Alkalosis may develop as a result of: (1) *Hyperventilation of the lungs* caused by forced breathing, whereby an excessive amount of carbonic acid is removed by the lungs Forced breathing may be self-induced, it is also seen in hysteria, in certain lesions of the brain and often in young infants by excessive crying. (2) *Excessive vomiting whereby large quantities of hydrochloric acid and sodium chloride are lost.* (3) The excessive administration of bicarbonate of soda or other alkalies, which may overbalance the hydrogen-ion concentration of the blood, causing an increase in the hydroxyl ion, that is, an increase in the alkalinity of the blood.

The clinical signs of alkalosis are: Slow, shallow, often irregular breathing (an increase in depth and frequency of the respiration may often remedy the alkalosis); cyanosis, and at times tetany or muscle cramp, tingling in the fingers, slight numbness of the extremities, some mental disturbance and, in severe cases, carpopedal spasm and general convulsions with the presence of various signs of tetany (See: Tetany, p. 790).

Sulfanilamide, Sulfapyridine, Sulfadiazine and Sulfathiazole Concentration in the Blood

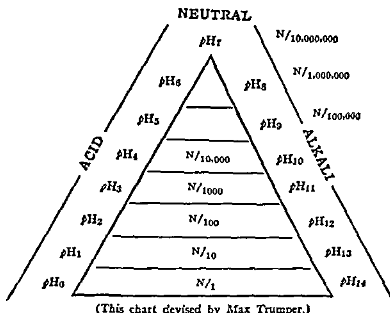
Sulfanilamide and its allied compounds have become common and frequently used remedies in a host of conditions. Because of their toxicity it is important to determine the concentration levels of these remedies in the blood after they have been administered for longer than 24 hours. Some patients will show a high concentration with comparatively moderate doses, while others will show a low blood concentration level with large doses. Since these drugs are toxic to sensitive persons, and may cause serious blood changes and *kidney complications, it is important* that the blood levels be checked frequently.

In mild or moderate infections a blood level of five to ten mg. per cent is considered desirable. In severe infections,

when mild symptoms may be apparent, *40 to 30 per cent; and in severe acidosis,* which gives rise to symptoms of intoxication, below 30 per cent.

III. ***Alkali Tolerance of Patient:*** Sellards[1] *states that the alkali tolerance* method is reliable for *proving* the absence of acidosis, but may not be entirely dependable *for demonstrating its pres*ence or the degree of acidosis when present. *This is probably* due to the fact that conditions that produce acidosis so influence the kidneys that the excretion of alkalies is markedly impaired. This test is simple, and is carried out in the following way:

Sodium bicarbonate is administered in small amounts, *either by mouth or intra*venously, until the reaction of urine changes from acid to alkali. The amount required for this change is noted; normally, the administration of from 5 to 10 *grams of bicarbonate of soda* by mouth is sufficient to produce an alkali reaction in the urine. Patients suffering from acidosis require a greater amount of *bicarbonate of soda to neutralize their* urine. The generally accepted method is to give *half* a gram of bicarbonate of soda per kilogram of body weight That should produce an alkali reaction of the urine in a normal person. The amount of bicarbonate of soda necessary to neu*tralize the urine in excess of a half a* gram per kilogram of body weight may indicate the degree of acidosis. Van Slyke's advice is to be careful not to use an excess of sodium bicarbonate to avert the danger of tetany.



(This chart devised by Max Trumper.)

IV. ***Carbon Dioxide Tension of the Alveolar Air:*** The method of determining the carbon dioxide tension is

[1] Sellards: Johns Hopkins Hospital Bulletin, 23-28, 1912.

phore which has a ferment-like action and the presence of (*a*) *agglutinins*, (*b*) *precipitins*, and (*c*) *opsonins* These are utilized for specific diagnostic tests.

(*a*) ***The Agglutinins:*** These have the property of agglutinating the type of microörganisms that are responsible for the development of the immunity toward the disease caused by them. The Widal reaction depends upon the agglutination or clumping of the typhoid and paratyphoid bacilli when they are brought in contact with the serum of an individual having typhoid or paratyphoid fever or one who recently had one of these diseases or was recently immunized against them Agglutination tests are therefore based upon the ability of the blood serum containing specific agglutinins to react against the particular organism causing the disease (SEE: p. 1062).

Agglutination tests are of two types (1) For the diagnosis of disease, where an unknown serum, that is, the serum of the patient whose diagnosis is sought, is brought into contact with a known organism as in the Widal reaction, and (2) for the identification of bacteria, where the serum known to contain specific agglutinins for one organism is brought into contact with a suspension of unknown bacilli. The clumping of the organism in high dilutions of the serum in a specified time identifies the disease in the one instance and the bacteria in the other. (Dilutions of not less than 1:80 in two hours or less) The agglutination test is employed clinically for the diagnosis of typhoid and paratyphoid fever, tularemia, undulant fever, epidemic meningitis, Asiatic cholera, bacillary dysentery, the plague, and occasionally for the various types of pneumococci, the Rickettsia diseases, and others The serum of patients suffering from some Rickettsia diseases, such as typhus fever, trench fever, and Rocky Mountain spotted fever agglutinate the bacillus Proteus X19, an apparently nonpathogenic organism found in the urine of those suffering from these diseases (Weil-Felix reaction).

(*b*) ***The Precipitins:*** These are employed for the biologic identification of unknown proteins, such as for the differentiation of human from animal blood in Forensic Medicine and for differentiating horse flesh from beef. In Clinical Medicine, it is employed for the diagnosis of echinococcus disease and for determining the types of the pneumococcus taken from the peritoneal washings of a mouse when there is contamination by other organisms.

The test for echinococcus disease is performed by mixing in a test tube, equal parts of the fluid from the hydatid cyst with the blood serum of the patient. This is permitted to stand for one-half hour. The appearance of a flocculent precipitate indicates a positive reaction

(*c*) ***The Opsonins:*** These are substances found in the blood that have the property of preparing the bacteria in the blood for ingestion by the leukocytes That is, they stimulate phagocytosis and are somewhat specific. A specific opsonin seems to stimulate phygocytic action for each species of bacteria. There are also opsonins for other formed elements in the blood, *i. e*, red corpuscles, dyes and other foreign bodies.

The opsonic index is obtained by the following method: The patient's blood serum, a suspension of the specific microorganisms and a suspension of washed leukocytes are mixed in equal parts in a test tube. Another test tube is similarly prepared, but using a normal person's blood serum instead of the patient's. Both tubes are incubated. Then smears

levels up to 16 mg. per cent may at times be necessary. If the patient exhibits toxic symptoms, the blood concentration must be kept at a lower than the required level or the drug must be discontinued. In the presence of polyuria, or diarrhea, the blood concentration does not attain as high a level with the same dosage of the drug as it does in oliguria or in constipation. Among the toxic symptoms produced by these drugs are nausea, *vomiting, headache,* diarrhea, renal symptoms, skin rashes, fever, and nervous symptoms.

Serologic Tests (Serology)

The principles upon which serodiagnostic tests are based are the immunologic reactions in the blood. The body's defense mechanism against pathogenic microorganism is such that when these *organisms enter the body in sufficient* numbers to cause disease, there develop within the body certain substances which attempt to neutralize and to destroy both the organisms and the toxins they produce. These substances are known as antibodies or immune bodies. They are found in the body tissues and fluids during the active stages of the disease and for varying periods after recovery

Antibodies or immune bodies are *species specific; that is, when they are* formed because of a specific organism they are capable of protection only against that type of organism or the toxins produced by them In other words, when a person has had a certain infectious disease, he becomes immune only to that disease, or to a very similar one, *i. e.,* vaccinia and smallpox. But, having had smallpox, a person would not be protected against typhoid fever, *syphilis,* etc. The immunity may be temporary or lasting, and may be produced either by having had the disease. *or by having been artificially immunized* against the disease as by the administration of small doses of bacterial toxins, of killed or attenuated *organisms, or by* introducing into the body specific antitoxins. Immunity against disease is in part carried out by the various specific actions of the immune bodies developed in the blood as the result of specific diseases Because of their specific reactions, *the immune bodies are divided into three* groups, namely: Immune bodies of the first order; immune bodies of the second order, and immune bodies of the third order

The Immune Bodies of the First Order: These are antitoxins They have the ability to neutralize toxins that are produced during a diseased process and also have the ability to protect or to immunize an individual against the pro*pagation of specific types of organisms* or to neutralize their toxins To this classification belong the various antitoxins like diphtheria, tetanus, etc. *They* are employed in treatment and prophylaxis but are not utilized for diagnosis. While the antitoxins may be employed to determine the degree of natural or acquired immunity, an individual possesses, they are not employable as a diagnostic test of the disease. As an example: The Schick test will indicate whether a person is or is not relatively immune to diphtheria, but it is of no *value as a test to determine* the presence of diphtheria.

Immune Bodies of the Second Order: *The specific action of this group* depends upon the presence of a *zymo-*

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Immune Bodies of the Second Order: The specific action of this group depends upon the presence of a zymo-

or bound to the antigen by this specific amboceptor, and no complement will be left in a free state.

"(*b*) If the patient's serum does not contain the specific antibody to serve as a connecting link, the complement will remain unbound or free in the fluid.

"In either case there will be no visible change to show what has taken place, and it is necessary to add an indicator which will show whether the complement still remains free. This is found in the two specific elements of the hemolytic system, red blood corpuscles and hemolytic amboceptor. If free complement be present the hemolytic system is completed and the corpuscles will be hemolyzed. If, on the other hand, all available complement has been bound to the antigen by the antibody, then hemolysis cannot occur."

Complement Fixation Test for Syphilis (Wassermann)

In the Wassermann test for syphilis, the *antigen* is usually a cholesterinized and lecithinized alcoholic extract of heart muscle. (This is more sensitive than syphilitic material or the spirochete)

The *amboceptor* is the clear serum of the patient's blood devoid of corpuscles and heated to 56° C, or 133° F

The *complement* is the blood serum of a guinea pig To the proper proportions of the antigen, amboceptor and complement is added a definite amount of indicator which consists of sheep's red blood cells and their respective antibodies or amboceptor obtained from the blood of a rabbit that had previously been injected with sheep's corpuscles If the patient's blood is syphilitic, the reagin in the blood will unite with the antigen and bind the complement so that no hemolysis of the sheep's corpuscles takes place, the reagin in the syphilitic blood having in this case bound the syphilis antigen with the complement. If, on the other hand, the blood of the patient is not syphilitic, there is no reagin in the blood to bind the complement with the syphilitic antigen; therefore, the complement is free to hemolize the sheep's corpuscles. This reaction is characterized by the formation of hemolysis and indicates a negative reaction.

A positive Wassermann reaction is indicated by the complete absence of hemolysis of the sheep's corpuscle resulting in a clear fluid. This is designated as "positive plus four" Plus three, two or one reactions are graded according to the degree of hemolysis that takes place In other words, a nonhemolytic reaction (a clear fluid) is positive for syphilis and a very marked hemolytic reaction (a very turbid fluid) constitutes a negative Wassermann.

A "four plus" or a strongly positive reaction indicates syphilis; "three plus" or moderately positive of Kolmer may be accepted as positive, particularly in the presence of a positive history or clinical manifestations "Plus two" and "plus one" reactions are doubtful, requiring repetition of the tests. During the course of treatment for syphilis, a "plus one" or "plus two" indicates that the disease is still active A negative reaction does not necessarily exclude syphilis as it only means that the Wassermann reaction is negative In the presence of clinical manifestations or a positive history, a negative Wassermann should not be construed as the absence of syphilis Several successive negative reports in persons who have not had antisyphilitic treatment would indicate the absence of syphilis.

Syphilis may be considered cured when the Wassermann reaction or Kol-

are prepared from each, are stained and examined under an oil immersion lens. The number of leukocytes are counted in each specimen and also the number of bacteria in each of the leukocytes. The average number of bacteria per leukocyte is calculated; this determines the phagocytic index. The phagocytic index of the patient's blood divided by the normal blood gives the opsonic index. The normal blood is considered at 1 It will also be found that not only are there more bacteria in the patient's leukocytes, but that more of the patient's leukocytes have ingested bacteria than have the normal blood's leukocytes.

Immune Bodies of the Third Order: This reaction depends upon the "complement fixation phenomenon" of Bordet and Gengou.

Three substances are required for the production of the complement fixation tests: (*a*) *An Antigen*· This may be red blood cells or microorganisms; (*b*) *an amboceptor* or specific antibodies, and (*c*) *a complement* When these substances are brought together, one of two reactions may take place If the antigen is made up of red corpuscles, hemolysis will occur; and if the antigen is bacteria, bacteriolysis results

Application of the Principles of Bacteriolysis and Hemolysis:[1] "It is necessary to bear constantly in mind the three substances or 'bodies' which are concerned in bacteriolysis and in hemolysis and the part which each plays This may be outlined as follows:

BACTERIOLYTIC SYSTEM

Antigen (invading bacterium)	+	Bacteriolytic amboceptor (in serum of infected person)	+	Complement (in serum of any normal animal)	=	Bacteriolysis

HEMOLYTIC SYSTEM

Antigen (red blood corpuscles)	+	Hemolytic amboceptor (in serum of animal injected with red corpuscles)	+	Complement (same as in bacteriolytic system)	=	Hemolysis

"The important fact in the above formulae is that, while antigen and amboceptor differ in the two systems, the complement is the same. Whatever the source of the complement, it will serve either for bacteriolysis or for hemolysis, and this is the key to the complement-fixation tests.

"In the application of these principles it is possible so to adjust the test that any two members of a system being known, the third may be determined qualitatively and (roughly) quantitatively In the clinical use of the test, however, one seeks the amboceptor whose presence in the patient's serum establishes the diagnosis of the corresponding disease. To accomplish this, one mixes in a test tube appropriate amounts of a culture or extract of the invading organism, blood serum from a suspected patient, and complement. One of two things will occur:

"(*a*) If the patient suffers from the disease in question and his blood serum, therefore, contains the corresponding amboceptor, the complement will be fixed

[1] Todd, J. C. and Sanford, A H: Clinical Diagnosis by Laboratory Methods, p 618, W B Saunders, 1939

CHAPTER XXXV

Exudates, Transudates and Body Fluids

The Cerebrospinal Fluid

In the presence of symptoms referable to the cerebrospinal system, a spinal puncture should be performed, for diagnostic purposes, and the spinal fluid *should be examined macroscopically*, microscopically and chemically. Spinal puncture is also employed as a therapeutic *measure to relieve intracranial pressure* and for the administration of sera and spinal anesthetics

Technic for Spinal Puncture: If the patient is not too ill to sit up, the operation may be performed in the sitting posture leaning well forward A sick patient should lie on one side, the thighs well drawn up upon the abdomen, the legs flexed, and the body bent as far forward as possible To maintain this flexed position of the spine, in the absence of adequate assistants, a large towel or a sheet may be passed over one shoulder and *under the knees and securely* held in place so that extension of the spine is impossible

The site of puncture is, as a *rule*, the fourth lumbar interspace, a line drawn posteriorly from one anterior-superior spine of the ilium to the other will cross this interspace. After the skin has been *thoroughly prepared*, the examiner chooses a spinal needle which is not too brittle, which measures 5 to 10 cm. in *length* and 1 *to 2 mm in* diameter, *and is* provided with a stylet In hypersensitive patients it is best to employ local anesthesia so as to minimize the discomfort. The needle is grasped near its point, and is inserted with steady pressure between the spines of the fourth and fifth lumbar vertebrae. When the sense of resistance suddenly ceases, the stylet is removed, and the fluid is permitted to flow through the needle. It is important to note the rapidity with which the fluid flows because in the absence of a spinal monometer this is an indication of the



Fig 1—Technic for spinal puncture.

degree of *intraspinal pressure*. Normally, the fluid flows at the rate of approximately one drop a second. When *the drops come rapidly, it indicates* increased pressure; when the stream is continuous, it is an indication of very *high pressure The pressure* can be accurately gauged only by an apparatus designed for the purpose. The normal *pressure is usually considered to be* between 100 and 200 millimeters of distilled water or 7 to 10 millimeters of mercury, and is *physiologically increased* by crying, coughing or muscular resistance during the operation.

mer's modification thereof and the Kahn test remain negative for several years after treatment is stopped.

Complement Fixation Test for Gonorrhea

This test is of greatest importance in cases of gonorrheal arthritis, as positive results may be obtained in about 80 per cent of cases. In acute gonorrhea, only 35 per cent are positive. In doubtful cases of arthritis, a complement fixation test for gonorrhea should be made and if found positive a diagnosis of gonorrheal arthritis may be made A negative report does not entirely exclude the specific origin of the disease. The test becomes negative in from two to four weeks after a cure is effected.

Complement Fixation Test for Tuberculosis

The technic of this test is similar to that of syphilis or gonorrhea. The value of this test in tuberculosis is questionable. While a great number of tuberculous patients may give a positive reaction, there are many nontuberculous individuals who also give a positive reaction and many cases of far advanced tuberculosis who react negatively.

Flocculation Test or the Precipitation Reaction for Syphilis

This is based upon the appearance of a white precipitate when an alcoholic extract of normal heart muscle is added to the blood serum of a syphilitic individual. This reaction differs from the Wassermann reaction where a positive is indicated by a clear fluid and a negative by hemolysis. The Kahn test is the most widely employed of this group, and is used as a control on the complement-fixation test. At times the Kahn test may be positive when the Wassermann reaction is negative, or the reverse may also occur.

Other tests for syphilis are the Kline test, the Eagle test, the Hinton test, the Meinicke test, the Sachs-George test.

These tests are also positive in yaws and in some of the other spirochetal infections.

Occasionally a positive Wassermann reaction may be found during the early stages of lymphogranuloma inguinale, and during some of the acute infections

Rivalta Test for Globulin

The Rivalta test is for the qualitative determination of the globulin fraction in an albuminous fluid Place a quantity of fluid to be tested in a test tube, add slowly a few drops (drop by drop) of a 0 5 per cent acetic acid solution. In the presence of globulin, a bluish smoky precipitate will appear in the test tube. When the quantity of globulin is fairly large, such as is found in an exudate, the bluish cloud will slowly settle to the bottom. In a transudate, because of its minute globulin content, the light bluish or smoky precipitate remains suspended in the fluid.

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Cerebrospinal Fluid Changes in Diseases of the Central Nervous System
(Modified after Kolmer and Boerner, "Approved Laboratory Technic," D. Appleton & Co., 1931)

Disease	Pressure	Type	Coag.	Cells	Qual Glob.	Qual. Alb	Quant Prot.	Quant Sugar	Chlor	Bact	Wass.	Gold
Normal	100–200	Clear and colorless	0	0–10 lymph	0	0	15 to 40	15 to 60	720 to 750	0	Neg.	Neg.
Serous Meningitis	Increased	Normal	0	Normal	0	0	Norm or sl. incr.	Norm.	Norm	0	Neg.	Neg
Anterior Poliomyelitis	Increased	Normal or opalesc	Fibrin web	0 to 2000 early poly later lymph	+	++	40 to 500	40 to 120	Norm	0	Neg	Meningitic or Zone II
Purulent Meningitis	Marked increase	Cloudy	Coag	100 to 5000 poly	++++	++++	Incr	0 to 60	Norm or decr	+	Neg.	Meningitic curve
Chronic bas. Meningitis	Normal or increased	Normal or opalesc	Coag	10 to 1000 poly	++++	++++	100 to 1000	20 to 60	Norm	0 or meningococci	Neg	Meningitic curve
Tuberculous Meningitis	Increased	Clear to Turbid	Fibrin web	80 to 1000 lymph	++ to ++++	++ to ++++	100 to 1000	0 to 40	500 to 700	t b.	Neg.	Meningitic curve
Epidemic Encephalitis	Normal or increased	Normal	Fibrin occ	10 to 200 lymph	± to +	+ to ++	30 to 200	40 to 120	Norm.	0	Neg	Neg or Zone II
Brain Pituitary and Pineal Tumor	Variable	Normal	0	10 to 80 lymph	± to +	+ to ++	20 to 200	40 to 100	Norm	0	Neg	Neg.
Intraspinal Tumor	Variable	Normal	Coag	Normal to 50 lymph	+ to ++++	+ to ++++	60 to 1000	Norm	Norm	0	Neg	Neg
Syphilis (1st and 2nd stages)	Normal	Normal	0	8 to 98 lymph	± to +	+ to ++	20 to 60	Norm	Norm	0	Var.	Neg or Zone II
Syphilis (Meningovascular)	Normal or sl incr.	Normal	0	2 to 1000 60% lymph	+ to ++	++ to +++	30 to 150	Norm or less	Norm	0	Pos	Luetic Zone II
Syphilis (tabes dorsalis)	Normal	Normal	0	10 to 75 lymph	± to +	+ to ++	30 to 60	Norm or less	Norm.	0	Pos 70%	Leutic Zone II
Syphilis (Paresis)	Normal or sl incr.	Normal	Coag	30 to 200 lymph	+ to ++++	++ to +++	50 to 100	Norm or less	Norm	0	Pos	Paretic Zone I
Multiple Sclerosis	Normal or sl incr	Normal	0	0 to 40 lymph.	+ to ++	+ to ++	20 to 80	Norm	Norm	0	Neg	50% neg Zone I or II may

Thoracentesis

Tapping of the chest may be performed for one of four reasons *First*—Actually to determine the presence of fluid in the pleura (*exploratory puncture*); *second*—to determine the character of the fluid; *third*—to withdraw the fluid from the serous sac, and *fourth*—for the introduction of air into the pleural sac (artificial pneumothorax).



Fig. 2—Technic for entering pleural cavity for withdrawing of fluid or performing artificial pneumothorax

Technic: The skin is scrubbed with soap and water, dried, and painted with tincture of iodine, which is then removed with alcohol A few drops of a one or two per cent solution of cocaine; novocain, or any other local anesthetic are injected into the skin at the site of the operation, and the hypodermic needle then pushed through the skin, so that the track is also anesthetized An exploratory needle attached to a 5 or 10 cc syringe is inserted in the interspace previously anesthetized.

The exploratory needle should hug the upper surface of the rib, thus avoiding injury to the subcostal vessel. When the fluid is removed, the macroscopic appearance will indicate whether it is clear, turbid or bloody. If the fluid is clear, it may be either an exudate or a transudate. A *transudate* is characterized by low specific gravity, traces of albumin and very few cells, while an *exudate* is an inflammatory product and therefore contains many cells, large quantities of albumin and is of high specific gravity When a large quantity of fluid is to be removed, the needle is attached to a "vacuum bottle," which draws off the fluid

Pericardial Puncture

The site for tapping the pericardium is usually the fourth intercostal space, close to the left edge of the sternum When the dullness extends a distance to the right of the sternum, and the apex beat is not displaced beyond the midclavicular line, a puncture may be performed in the fourth or fifth intercostal space, to the right of the sternum. The pericardial fluid may be clear (transudate), or somewhat turbid and of high specific gravity (exudate); and may contain pus

Significance of Aspirated Fluid: A *transudate* (clear fluid) may be found in the pleural cavities as the result of heart failure, of compression of the lungs or of a vein in the chest by tumors, aneurysm, etc; it is also found in nephritis, particularly in the type with water retention, and in grave anemia. Transudates into the pericardium may be found in severe myocarditis and in general anasarca.

Exudates are usually found as a result of inflammatory processes such as pleurisy and pneumonia, and may also be found in acute and chronic pericarditis

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Multiple Sclerosis	Normal or sl. incr	Normal	0	0 to 40 lymph	+ to ++	+ to ++	20 to 80	Norm	Norm	0	Neg	50% neg Zone I or II may

meal, has been taken. The gastric contents may be withdrawn as a single procedure with a large stomach tube or pump, a definite number of hours after a given meal or, preferably, may be withdrawn with the Rehfuss tube every 15 minutes over a period of two hours or more. The latter procedure is known as the *fractional method*. The gastric juice upon a chart. Fig. 2 is an example of a compact and serviceable chart for recording the results of a gastric analysis and examination

Kind of Food: Starches usually cause less hydrochloric acid to be secreted in the stomach than do meats, as *proteins demand large quantities of hydrochloric acid for digestion*. Various

GASTRIC ANALYSIS DATE

	F	20	40	60	80	100	120			ACID									
Amt										150									
Food										135									
Color										120									
Mucus										105									
Bile										90									
Blood										75									
Free										60									
Total										45									
Rennet										30									
ORG Ac										15									
Oc Bl										0									
TIME											F	20	40	60	80	100	120		

Microscopy

Saliva Retching Stomach empty in hrs

Secretion Motility

Curve Spasm

Interpretation

Fig 3—Chart for plotting gastric analysis

is normally secreted during the process of digestion In its pure state it contains two per cent HCl (pH 1 7), is almost odorless, is pale yellow in color, and has a specific gravity of 1 002 to 1 005 Very often the kind of food ingested alters the appearance of the gastric juice Its composition also varies greatly with the kind of food ingested, the length of time digestion has been in progress, and the peculiarities of the individual under examination The various gastric findings may be graphically recorded drugs, such as alcohol, tobacco and strychnine, also have a distinct effect upon the outpouring of gastric juice

Time of Digestion: From the time food enters the stomach until it is completely digested, the quantity of gastric juice secreted varies in accordance with the amount of work it has to perform, thus, when a small quantity of food is ingested, little gastric juice is required for its digestion, but a heavy meal demands a large amount of gastric juice. The height of the digestive stage usually

of rheumatic or tuberculous origin. Pus in the pleura (empyema) results from some suppurative process affecting the lungs and pleura In tuberculosis and after pneumonia, empyema is fairly common. Often an ordinary pleural effusion may become infected by pyogenic organisms, thus changing a pleurisy into an empyema. *Pyopericarditis*, or pus in the pericardial sac, results from pyogenic infection of the pericardial fluid It may be secondary to myo- or endocarditis, or may result from a stab wound.

Bloody Fluid: The presence of bloody fluid, if not caused by a direct injury, may be due to neoplasm of the lung, most frequently carcinoma, at times to tuberculosis of the lungs with infection of the mediastinal glands, and to aortic aneurysm, ruptured varicosities, purpura and other hemorrhagic diseases.

Paracentesis Abdominis

The skin of the abdomen is prepared in the usual surgical manner, and the patient sits on the edge of a chair or bed, the feet supported and the knees widely separated. (The bladder must be emptied before an attempt is made to tap the abdomen.) The lower portion of the abdomen, just above the symphysis pubis in the midline, is chosen as the site for tapping. The skin at that point is anesthetized with a local anesthetic, the skin is incised with a sharp scalpel, and a trocar-cannula quickly thrust through the open wound The trocar is removed, and the fluid permitted to flow through the cannula.

Significance of Abdominal Fluid (ascitic fluid): The fluid of ascites may be clear, turbid or bloody, and either white or yellow in color. *Clear fluid* or *transudate* usually results from heart failure, disease of the kidneys, severe anemia, liver disease, such as atrophic cirrhosis, or from compression of the inferior vena cava.

Exudates are due to some inflammatory process, and may be found in association with inflammatory conditions of the liver, omentum and bowel. *Pus* is usually found with severe inflammatory conditions of the peritoneum, resulting from a ruptured appendix or gallbladder, abscess of the liver or spleen, or a ruptured pus tube. *White fluid*, not pus, is usually chyle, caused by compression of the chyle duct. *Yellow-stained fluid* may be found in association with jaundice, or after direct injury to the gallbladder, common bile duct or pancreatic duct

An *ovarian cyst*, when extremely large, may sometimes be mistaken for ascites On tapping, the fluid obtained from an ovarian cyst is usually of a high specific gravity (1 030), and of a dark brown grumous appearance. Microscopically, a large number of round or oval cells, showing dense irregular granulation often obscuring the nucleus of the cell, will be found

Hydatid cyst is recognized by the presence of hooklets, scolices and cyst membranes, the fluid is almost colorless and of very low specific gravity, containing little or no albumin.

Gastric Contents

Stomach conditions cannot be definitely diagnosed by gastric findings alone, because gastric findings are not distinctly characteristic of a definite condition. The most that can be said is that certain conditions may produce a gastric juice which is more or less characteristic.

The gastric juice may be extracted from a fasting stomach or after a definite quantity of food, known as a *test*

during the course of certain fevers, or in mucous gastritis, carcinoma of the stomach and pernicious anemia Reliance cannot be put upon the evidence obtained from gastric analysis alone; it only aids in establishing a diagnosis when combined with other methods of clinical, physical and radiological examinations

The absence of free hydrochloric acid after the histamine test points strongly to carcinoma of the stomach or pernicious anemia.

Blood: Blood in the stomach contents, either microscopic or macroscopic, may result from trauma to the esophagus or gastric mucosa by the swallowing of the stomach tube, or may find its way into the stomach from lesions in other organs. When extragastric causes and direct injury can be eliminated, the commonest causes for blood in the gastric contents are *ulcer* and *carcinoma* of the stomach (SEE · pp 641 and 643)

The presence of Boas-Oppler bacilli is an indication of malignancy.

Mucus: A moderate quantity of mucus is constantly found in the stomach and acts as a protection to its mucosa against hot and irritating substances An absence of mucus is often found in hyperchlorhydria and gastric ulcer, while an increased amount of mucus in the gastric contents indicates catarrhal gastritis, this may also occur in carcinoma

Fatty Acids· Fatty acids, lactic and butyric, are abnormal constituents of the gastric contents. When neither of these acids has been ingested, their presence in the gastric contents often indicates malignant disease of the stomach

Bile: Bile may be found in the stomach contents if regurgitation through the pylorus has occurred; this is often found in gallbladder disease, duodenitis, and in conditions causing a patulous pylorus.

The Feces

Characteristics of Normal and Abnormal Stools

In many diseases it is important to make a macroscopic and microscopic examination of the feces. Occasionally a disease can be diagnosed only by a microscopic examination of the feces; for instance, in the search for the cause of a chronic diarrhea the presence of *Endamoeba histolytica* definitely establishes the diagnosis. In certain obscure anemias the finding of ova and parasites will often greatly aid in making a proper diagnosis In a macroscopic examination of the stool the odor, color and consistency, the presence of blood and parasites should be especially observed. In the microscopic examination, bacteria, fungi, parasites, ova, blood and pus cells, and the variety of food remnants should be noted

The *normal stool* is semisolid, usually formed, has a characteristic odor and is of yellowish brown color. *Pathologically,* the stool may be altered in shape, consistency and color.

Pathological Alterations: (*a*) ***Shape:*** Ribbon-shaped stools are found in conditions that cause constrictions of the anus, cancer or stricture of the rectum, ischiorectal abscess, enlarged prostate, large hemorrhoids, and spasmodic contraction of the rectum. At times uterine tumors or a large prolapsed uterus may cause ribbon-shaped stools.

(*b*) ***Consistency:*** Semifluid or fluid fecal matter is found in all cases of diarrhea, whether due to acute gastrointestinal disturbance or to the administration of purgatives, particularly the salines. It is also found in tuberculosis, typhoid fever, bacillary dysentery, amebic dysen-

coincides with the period in which the greatest amount of gastric juice is thrown out. This period in turn depends upon the *kind of food ingested; thus,* after a light test breakfast consisting of a roll, or a slice of bread weighing two or three ounces, and a cup of weak tea, the height of digestion will be reached within about one hour. A *test meal* consisting of a tablespoonful of barley gruel will reach its height of digestion within about two hours; while a test dinner consisting of meat, vegetables and soup will not reach its height of digestion for three or four hours. When the gastric contents are withdrawn from the stomach *in one procedure,* the withdrawal is performed at the end of one, two or four hours, depending upon the particular kind of test meal ingested. When the *fractional analysis* is made, a portion of the stomach contents is withdrawn in the fasting state, that is, just before the test meal is given. With the tube still in the stomach the test meal is eaten; 15 minutes later the second specimen is withdrawn. This is continued every 15 minutes until the stomach is empty The stomach contents thus obtained are examined, the result indicating the quantity and quality of the gastric juice secreted during the various stages of digestion. When the gastric contents are studied one must bear in mind the kind of a test meal employed

Quantity of Gastric Juice (test breakfast of Ewald): The quantity of filtrate obtained one hour after this test meal should vary from 30 to 50 cc. An increase in quantity may be due to acute or chronic hypersecretion (gastrosuccorrhea), or to gastric retention, the result of pyloric obstruction, gastrectasis, etc. Gastric juice is also increased in gastric neurosis. A *diminished* quantity may be caused by sudden fright, chronic gastritis, atrophy of the mucous membrane, hypertonicity of the stomach, or by an excessive amount of mucus in the stomach. Absence of free HCl in the gastric juice is found in achylia gastrica, in carcinoma of the stomach, pernicious anemia, grave secondary anemia, chronic gastritis, and is often a result of atrophy of the gastric mucous membrane.

Total Acidity: One hour after the test meal is swallowed, the total acidity varies from 50 to 60. By total acidity is meant the amount of free and combined hydrochloric acid. When the total acidity rises from 60 to 100, it may be caused by increased ingestion of acid, if over 100, the condition is considered *gastrosuccorrhea* (hyperchlorhydria), if under 30, it is considered *hypochlorhydria.* Absence of acidity, particularly when associated with the absence of ferments, is known as *achylia gastrica.*

Free Hydrochloric Acid: The normal quantity is about half of the total acidity. Free hydrochloric acid appears in the gastric contents after the basic affinities have been satisfied. During the early stages of digestion, the hydrochloric acid secreted combines with the albumoid and basic substances of the gastric contents to form soluble albuminous substances The quantity secreted above that required for this purpose is known as free hydrochloric acid

An increase in the free and combined hydrochloric acid is usually found in nervous gastritis, irritation of the stomach, gastric and duodenal ulcer, pyloric stenosis, secondary irritation and congestion due to a gallbladder disease, spastic colon and chronic appendicitis; also in vagotonia.

Diminished or absent free hydrochloric acid may be due to the too early evacuation of the stomach contents, as

stool, indicating a lack of bile. A musty *or mousy odor is usually found in cholera infantum.* Stools having the odor of sulfurated hydrogen are found after the administration of sulfur and the eating of large quantities of eggs. The stools are also offensive in typhoid fever and in intestinal putrefaction.

*(e) **Abnormal Contents in the Stool:*** *Pus in the stool*, when found in large quantities, may be due to the rupture of an abscess into the alimentary canal. In small quantities, it is found in dysentery and in ulcerated conditions of the gastrointestinal canal. *Membranous shreds* are an indication of sloughing of the intestinal mucosa, and are found in acute proctitis, dysentery, ulcerative colitis, enterocolitis and gastroenteritis.

In addition to the above mentioned constituents of the stools, other material, such as *gallstones, parasites, seat worms, tapeworms, roundworms* and other *intestinal parasites* or their *ova* may be found. Whenever, in the absence of asthma, the blood shows an eosinophilia of over five per cent, the feces should be examined microscopically for parasites or ova. In the anemias the *feces should also be searched for parasites,* particularly tapeworms. In the presence of persistent diarrhea, the feces should be examined for microorganisms, parasites or their ova.

Fatty stools are found in association with disease of the pancreas, such as carcinoma, chronic pancreatitis, also as a result of great indulgence in fats and oils.

Gases in stool occur as globules of various size. When small or pinpoint it indicates putrefaction. When large and globular it is indicative of fermentation and such stool usually floats.

Foreign bodies may appear in the stool *after being swallowed or because of their* introduction into the rectum. Young children and insane patients are apt to swallow foreign bodies.

The Sputum

The secretions and excretions of the respiratory tract, excluding those of the nasal mucosa, when brought up in large or small quantities, are known as *sputum*. When a specimen is to be examined, one must be certain that the material brought up is from the bronchi or lungs, and not the saliva from the mouth, or excretion from the schneiderian membrane. Sputum is examined both *macroscopically* and *microscopically*.

Macroscopic Appearance of the Sputum

The general appearance of the sputum depends upon the underlying conditions which cause expectoration.

(*a*) **Quantity:** The quantity may be large, so that a very light cough or only "hacking," and, at times, even a change of posture, will bring up expectoration. Again, the expectoration may be so scanty that repeated efforts at coughing will bring up but a minute quantity. At times, sputum may be absent, because the person coughing swallows it. As a general rule it may be stated that all *acute inflammatory* conditions of the pulmonary-bronchial system are attended with *scanty* expectoration; *e. g.*, in acute bronchitis, in the early stages of lobar pneumonia, and the beginning of bronchopneumonia.

In *chronic inflammatory* conditions of the pulmonary-bronchial system the expectoration is *copious, e. g.,* in chronic ulcerative tuberculosis of the lungs; chronic bronchitis (with or without

tery, or as a result of any inflammatory lesion in the large or small intestine.

Serous stool is composed of fluid without fecal matter; such stool is found in Asiatic cholera, cholera morbus, cholera infantum, poisoning by arsenic, antimony, toadstools, or any other poisonous foods, and at times in the presence of cancer of the lower portion of the rectum, and in such lesions of the intestinal walls as may be caused by parasites, ameba, and various bacilli Deep lesions of the gastrointestinal mucosa due to any cause may produce serous diarrhea

Mucous stool is usually indicative of mechanical or pathologic irritation of the bowel. Small particles of mucus upon the surface of the stool are often found in health Large quantities of mucus either mixed with the stool or covering the stool is found in disease of the intestinal mucosa and in most types of colitis, also in acute intestinal obstruction, foreign bodies, malignancy of the colon or rectum and impacted feces. Mucous diarrhea is found in dysentery, mucous colitis and proctitis.

Diarrhea of nervous origin is often found as a result of sudden emotions, such as fright, hilarity and anxiety.

Diarrhea of digestive origin is found after intoxication by food or drink, overeating, ingestion of an unusual quantity of fat, also in the presence of increased secretion of bile, atrophic cirrhosis of the liver and cardiorenal and vascular disease.

(*c*) ***Color:*** This often varies with the kind of food ingested; thus blackberries or blueberries will impart a dark color to the stool, while an exclusively milk diet will make the stool yellow. *Clay-colored stools* occur with obstructive jaundice.

The *green* stools of infants may be caused by intestinal fermentation, the ingestion of certain drugs (calomel) or by an unusual amount of bile being thrown into the intestines, and occur as a result of some forms of acute and chronic diarrhea.

Black stools may be caused by ingestion of bismuth, iron and manganese.

"Tarry" stools indicate digested blood, the hemorrhage being high up in the gastrointestinal tract. This is found in some cases of duodenal ulcer, gastric ulcer, or ulcer anywhere within the small intestines, also in melena neonatorum Tarry stool may also occur with carcinoma of the stomach or of the esophagus, purpura hemorrhagica, hemophilia, leukemia, dysentery, swallowing of blood from bleeding gums, tonsils, nose or other bleeding points, and the swallowing of blood from hemoptysis.

Bloody Stools. Bright red blood when seen in the stool often indicates that the bleeding originates in the lower bowel. It may be due to hemorrhoids, rectal fissures, carcinoma or polypi of the rectum, or to any inflammatory condition of the sigmoid and rectum associated with bleeding, or with acute congestion as in strangulated hernia, and other forms of acute intestinal obstruction The severe anemias, portal thrombosis, pernicious malaria, yellow fever, acute yellow atrophy of the liver, poisoning by mercury, lead and other poisonous substances should be enumerated among the causes that produce bloody stools.

(*d*) ***Odor:*** An unusually offensive odor of the stools is found in syphilic ulceration and gumma of the bowel or rectum, and in other ulceration of the rectum, also in gangrenous dysentery. An offensive odor, not putrid, will be noticed in association with clay-colored

SEMEIOLOGY OF THE SPUTUM

Macroscopic Appearance	Microscopic Features	Clinical Significance	Associated Clinical Signs
SEROUS—Appearance: Frothy, colorless, mucilaginous			
	Fibrin Eosinophile cells Alveolar epithelium Sometimes spiral strands (Curschmann's spirals)	**Chronic serous bronchitis.**	Those of chronic bronchitis
SEROMUCILAGINOUS—Appearance: Like syrup of acacia or whipped cream, sometimes slightly tinted with pink (albuminoid)			
See illustration above [Serous]	Fibrin Eosinophile cells Alveolar epithelium Sometimes spiral strands See illustration above [Serous]	(*a*) After too deep or too rapid **thoracentesis.** (*b*) Acute pulmonary edema in chronic nephritis, aortic disease, high pressure cases, heart cases, and pregnant women	(*a*) Those of acute edema. Violent dyspnea. Frequent cough Cyanosis Fine rales spreading through the chest. (*b*) Those of the causative disease Albuminuria, high blood pressure, pregnancy, etc Stethoscopic signs

emphysema); bronchiectasis, gangrene of the lungs, edema of the lungs and bronchial blennorrhea.

(*b*) **Consistency:** This depends largely upon the quantity. *As a general rule, sputum which occurs in large quantities is more fluid than when it is scanty.*

(*c*) **Color:** This varies with the amount of decomposition, the origin and the cause of expectoration. *Mucoid or glassy* sputum is practically colorless, somewhat viscid, resembling egg albumen or saliva. A *greenish* or *yellowish* tinge to the expectoration usually depends upon the amount of cellular elements entering into its formation. When decomposition occurs, the sputum usually assumes a yellowish green color *Bloody sputum* occurs in conjunction with small or large hemorrhages Pure blood intimately mixed with sputum indicates hemorrhage in the finer bronchioles, or that blood from other sources has been mixed with sputum.

Streaked sputum (where streaks of blood run through an otherwise unstained sputum) is found in conditions where minute hemorrhages occur in the upper air passages or in the mouth. Pure blood is expectorated because of rupture of a large vessel, an aneurysm or several abrasions of small vessels *When the blood is brought up in large quantities (hemoptysis) it indicates that a large vessel has been ruptured; when the* blood is expectorated in small quantities, the lesion is in the smaller vessels; this is seen in pulmonary tuberculosis, mitral stenosis, and varicosities of the bronchi.

Rusty sputum means an admixture of blood and sputum so intimate as to give it that appearance, and is seen in lobar pneumonia, also in acute pneumonic phthisis.

"Anchovy sauce sputum" is also a result of intimate mixture of blood and sputum. It is dark brown in color and usually indicates partial decomposition Such sputum may be obtained from gangrene of the lung, abscess cavity and at times because of the rupture of an amebic abscess of the liver into a bronchus

Sputum resembling raspberry or currant jelly is at times observed in cases of tumor of the lungs.

(*d*) **Odor:** The sputum usually has a musty odor.

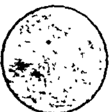
Fetid sputum is an indication of putrefaction of the lung, and is seen in cases of pulmonary abscess, gangrene, fetid bronchitis and at times also in bronchiectasis A *fruity odor* to the sputum, resembling that of stewed prunes, often precedes rupture of an echinococcus cyst.

Classification of the Sputum as to Its Physical Characteristics and Contents

Mucous sputum is either glassy, transparent, or of a whitish gray color, and of a light jellylike consistency. It is found in the first stages of acute bronchitis or tracheobronchitis The secretion of the bronchial mucosa is usually of that type.

Mucopurulent expectoration consists of a mixture of mucus and pus, is of a yellowish green color, and not transparent. It may be stringy or contain small particles of hard material. *It often assumes a flattened coin-shaped (nummular) appearance* when allowed to fall into a vessel filled with water. It may also contain small lumps or balls floating in the watery part of the sputum. When allowed to stand, it separates into three different layers: The upper layer consisting of small masses, the second layer of *slimy purulent material made up of*

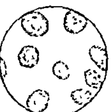
BLOODY.—**Appearance**: Blood *distinctly* predominates and is *more* or less pure, in greater *or less* amount, and with or *without* admixture of fibrin or mucopus

(Hemoptysis, *q v*) 	Red cells with or without admixture of 1 Mucin and fibrin 2 Pus corpuscles 3 Various bacteria (Tubercle bacilli in tuberculosis) 	(*a*) Disorders of the lung (1) ***Pulmonary* tuberculosis** 1 Incipient Initial symptom 2 Established Recrudescence 3 Cavities { Rupture of vessels / Ulceration (2) Cancer, gangrene, or cyst (*b*) Disorders of the circulation (1) **Mitral stenosis** (2) Aortic aneurysm (3) Heart failure	(*a*) The usual stethoscopic signs. *Constitutional* manifestations: Fever, impaired nutrition, anorexia, asthenia, sweats, etc Fluoroscopic evidences Tubercle bacilli in the sputum. (*b*) The usual signs of heart disease

PSEUDOMEMBRANOUS.—**Appearance:** False membrane, a bronchial cast of natural size

		(*a*) Branching and tubular false membranes (1) Diphtheritic or nondiphtheritic (2) Chronic pseudomembranous bronchitis (rare) (*b*) Whole or fragmented hydatid vesicles (hydatid cyst)	

PNEUMONOKONIOSES.—**Appearance.** *Mucopurulent*, this type of sputum requires separate mention because of its pigment content.—(*a*) Black streaks *Anthracosis* of coal workers, (*b*) *Red or iron* gray Siderosis of workers in *iron*, (*c*) Colorless, but sandy *Chalicosis* of stonecutters

	Alveolar cells containing pigment or dust particles 		

SEMEIOLOGY OF THE SPUTUM (*continued*)

Macroscopic Appearance	Microscopic Features	Clinical Significance	Associated Clinical Signs
PUTRID. FETID. GANGRENOUS.—**Appearance:** Fetid odor of gangrene, when set aside separates into 3 layers (*a*) Upper, mucopurulent; (*b*) middle, fluid, flocculent; (*c*) lower, greenish-brown			
	1 Unrecognizable polymorphonuclears 2 Mixture of bacteria	(*a*) 1st degree Temporarily putrid sputum: **Temporary fetid bronchitis**, a momentary complication of bronchiectasis (*b*) 2nd degree Constantly putrid sputum **Gangrene of the bronchi.** Curable gangrene of the lungs (bronchiectasis, cavities, etc.) (*c*) 3d degree Gangrenous sputum (putrefactive, necrotic, or fecal odor) **Gangrene of the lung.**	(*a*) and (*b*) **Physical signs** of chronic bronchitis with bronchiectasis Effect of posture on the cough and expectoration (*c*) Physical signs of softening of a lung focus or gangrenous empyema General health markedly impaired
RUSTY, FIBRINO-HEMATIC.—**Appearance:** Mucofibrinous and viscid, adhering to the receptacle, "rusty" or brick-red; currant or apricot jelly, exceptionally "prune juice" or "licorice juice."			
	1 Polymorphonuclears. 2 Red cells 3 Fibrin and mucin 4 Encapsulated pneumococci	(*a*) **Acute *lobar pneumonia*,** in 9 cases out of 10 (*b*) Rare causes (1) Cancer of the lung (2) Pulmonary infarction (embolism in heart cases) (3) Bloody sputum without infarction in heart cases or in albuminurics (4) Acute pleuropulmonary congestion due to influenza, etc. (pseudopneumonia)	(*a*) The usual evidences of pneumonia: Pain in side, auscultatory signs, and temperature indications (*b*) The signs of the causative disorder The sputum seldom presents a definitely rusty appearance, exhibiting features intermediate between rusty sputum and hemoptysis

watery mucus and serum, while the third layer is entirely confluent and contains decomposed pus. Such sputum is often found in chronic bronchitis, emphysema, pulmonary tuberculosis, and oftenest in bronchiectasis.

Purulent expectoration consists of almost pure pus; it may be seen in cases of gangrene of the lungs, rupture of a pulmonary abscess, rupture of an empyema or may represent the contents of pulmonary tuberculous cavities.

Serous expectoration consists of very thin fluid mixed with a small proportion of mucus and a small quantity of blood serum. It is found in cases of edema of the lungs.

Frothy expectoration is an admixture of air bubbles with serous fluid. It is found in edema of the lungs, after a spontaneous pneumothorax and, at times, in pulmonary emphysema.

Dittrich's plugs, yellowish white masses the size of a mustard seed, may be observed with the naked eye. Their presence indicates putrid bronchitis, pulmonary gangrene, or any other condition of the lungs that causes disintegration of pulmonary tissue.

Fibrinous Bronchial Casts At times a perfect cast of the inner lining of several bronchial ramifications may be found in the sputum, because of fibrinous bronchitis.

Curschmann's spirals are often found in the sputum of asthma and chronic bronchitis. These spirals are usually entangled with Charcot-Leyden crystals and numerous eosinophils.

Elastic fibers are found in the sputum in any condition that causes lung destruction, their presence even in the absence of tubercle bacilli strongly suggests tuberculosis. Often foreign bodies, such as hematoidin crystals, are found in the sputum of old pulmonary abscesses or perforated empyema. Crystals of *calcium phosphate* in the sputum usually indicate retention and stagnation. *Yellowish* or *grayish green* granules are often found in the sputum of pulmonary actinomycosis *Various parasites,* such as Trichomonas, Bilharzia, Ameba coli, taenia, Echinococcus, Ascarides, Actinomyces, and other parasites and fungi are often found in the sputum of sufferers from the conditions caused by them.

Bacteria in Sputum: ***Tubercle Bacilli:*** These are pathognomonic of tuberculosis of the lungs. When tubercle bacilli are persistently found in the sputum in large numbers, it is an indication of an active infection. When few in number, they indicate a not very active infection. The temporary absence of tubercle bacilli from the sputum of a person having signs of pulmonary tuberculosis should not be taken as proof positive against tuberculous infection of the lungs in that particular person, because the finding of tubercle bacilli in the sputum simply means that an open lesion exists, while the absence of tubercle bacilli from the sputum over a brief period may merely indicate that they are not being expectorated.

Pneumococci: Pure, or nearly pure, cultures of pneumococci are found in lobar pneumonia. To make a proper diagnosis, it is not sufficient merely to find pneumococci in the sputum The type to which that case belongs should also be determined The 30 or more types of pneumococci may be distinguished by the Neufeld method of typing the sputum (SEE: p. 1054).

Other Bacilli: *Influenza bacilli* may be found either in pure culture or in association with staphylococci, streptococci

Macroscopic Appearance	Microscopic Features	Clinical Significance	Associated Clinical Signs
MUCOUS.—Appearance: Clear, viscid, often air-laden fluid.—(*a*) Special variety Opalescent and highly cohesive, to the point of forming small ovoid or wormlike masses			
	1 Spiral strands (Curschmann's spirals) almost constantly present 2 Charcot-Robin (Charcot-Leyden) crystals	(*a*) First stage of **acute bronchitis.** (*b*) At the close of an asthmatic seizure (Laennec) (*c*) Fibrogummatous **syphilis** (softening) Round or ovoid masses of pea size in the mucoid fluid	(*a*) Rhonchi and sibilant râles (*b*) Attacks of asthma (see Asthma) (*c*) History Sluggish course of the disease Efficacy of antisyphilitic treatment
MUCOPURULENT.—Appearance: Yellow or greenish yellow mixture of mucus and pus, or purulent masses in frothy fluid			
	1 Mucin 2 A few epithelial cells 3. A few polymorphonuclears (Tubercle bacilli in tuberculosis)	(*a*) **Acute bronchitis** (second stage) (*b*) **Chronic and subacute bronchitis** (*c*) **Acute tuberculosis** (caseous pneumonia) (*d*) **Chronic pulmonary tuberculosis** (softening and cavities) (nummular sputum)	(*a*) and (*b*) Rhonchi and sibilant râles (*c*) Signs of apical pneumonia (*d*) Usual signs of apical consolidation, softening, and cavities
PURULENT.—Appearance: Pus, sometimes blood streaked, like pus from an abscess			
	1 Many polymorphonuclears, some showing karyolysis 2 Mucin 3 Streptococci, diplococci, cocci, or rods (Tubercle ba-	(*a*) **Cavities** (*q v*) (1) **Pleural** Empyema (general in ferlobar, or encysted) (2) **Pulmonary** Lung abscess, suppurating hydatid cyst (3) **Bronchial.** Bronchiectasis (4) **Subdiaphragmatic abscess** Hepatic, pyelonephritic, etc (*b*) **Pneumonia** in the stage of gray	(*a*) The signs of the causative disorder, plus 1 Sharp pain in the side Cough Dyspnea 2 More or less, copious vomiting of pus 3 Immediate relief from symptoms previously present

sufficient to fill the cavity) is brought up because of this change of posture.

Perforated Empyema: The sputum very much resembles that of pulmonary abscess.

Pneumonoconiosis: The sputum in this condition depends upon the kind and amount of dust inhaled Thus, in *anthracosis* (coal dust), the sputum is black, at least it contains black particles of coal.

Siderosis: The sputum resembles that of chronic bronchitis, and contains alveolar cells, and dark particles of iron and other metals.

Silicosis: In this condition the sputum contains particles of silica, or other stone dust.

Calcicosis: In this condition the sputum contains particles of lime and of plaster-of-Paris or other chalky deposits

Chemical Reaction of Sputum: Freshly expectorated material is usually of alkaline reaction, but turns acid on standing

or pneumococci. *Diphtheria bacilli* are often found during the course of this disease or in those who are diphtheria "carriers." Sputum containing *staphylococci, streptococci,* and *pneumococci, Friedländer's bacilli* and various microorganisms is observed in bronchopneumonia and in other acute or chronic respiratory diseases.

Chief Characteristics of Sputum in Various Conditions

Acute Bronchitis: During the early stages, the sputum is scanty, more or less transparent, but not viscid. As the disease progresses, the sputum becomes more copious, *is mucoid and may contain pyogenic microörganisms.*

Chronic Bronchitis: The expectoration is profuse, greenish yellow in color, mucopurulent and contains a profusion of bacteria.

Bronchopneumonia: During the early stages, the sputum is scanty, often *frothy, mucoid or mucopurulent. As the disease progresses,* the sputum becomes distinctly mucopurulent, is copious in amount and often contains blood, giving it a "prune-juice" appearance, it may also contain a variety of bacteria.

Lobar Pneumonia: During the early stages *the expectoration is scanty and viscid, yellowish in color,* somewhat mucopurulent and contains various types of pneumococci. Even in the later stages, particularly near or soon after the crisis, the sputum is viscid, tenacious and blood tinged, often being rusty in color.

Bronchial Asthma: *At first the sputum is scanty,* later it becomes purulent and grayish in color. It is as a rule frothy and contains Curschmann's spirals, Charcot-Leyden crystals and eosinophils.

Pulmonary Abscess: The quantity of sputum depends upon the amount of pus brought up from the abscess, and the *conditions of the lung tissue* surrounding it. The *sputum is usually purulent,* has a fetid odor and contains many pus cells, hematoidin crystals and portions of lung tissue.

Gangrene of the Lung and Putrid Bronchitis: The sputum is purulent, *has a most obnoxious odor, and, on standing, separates into three layers.* It contains pus cells, leukocytes and hematoidin crystals

Pulmonary Tuberculosis: In the early stages, before active consolidation has occurred, the sputum is scanty, gray*ish yellow or whitish in color.* It is frothy and is brought up in small quantities, often only as a spray during the act of coughing In the presence of consolidation, when not excessively large, the sputum becomes more copious, *is yellowish gray in color, and somewhat* tenacious In the late stages the sputum is mucopurulent, grayish yellow or yellow, has a musty and, at times, a fetid odor, contains fibers and tubercle bacilli *and not infrequently it may be blood*stained, blood tinged, or intimately mixed with blood. The expectoration of pure blood constitutes a hemoptysis (hemorrhage from the lungs).

Bronchiectasis: The sputum is *mucopurulent and when expectoration is infrequent,* the odor is foul The mode of expectoration is more or less characteristic, usually a patient suffering from bronchiectasis will bring up a very large quantity of mucoid *expectoration* at *infrequent periods* of the day, often merely as a result of change of posture At times a patient may not cough all day or night except on first arising in the *morning* when a large *quantity (accumulation*

sufficient to fill the cavity) is brought up because of this change of posture.

Perforated Empyema: The sputum very much resembles that of pulmonary abscess.

Pneumonoconiosis: The sputum in this condition depends upon the kind and amount of dust inhaled. Thus, in *anthracosis* (coal dust), the sputum is black, at least it contains black particles of coal.

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Chemical Reaction of Sputum: Freshly expectorated material is usually of alkaline reaction, but turns acid on standing.

CHAPTER XXXVI

Functional Tests

Function tests are carried out in order to determine the functional capacity of the organ under examination If the organ under examination is a secretory or excretory gland, its ability to secrete or excrete certain substances is investigated as to the quantity and the length of time required for the secretion or excretion of a definite substance. Thus the functional capacity of the kidneys is investigated as to their ability to concentrate various ingested products, dyes, water, salt, etc. The functional capacity of the liver is studied in order to gauge the activity of its various functions.

The functional capacity of the heart is studied by the electrocardiograph, the polygraph, the sphygmograph and some of the various exercise tests, and the peripheral vascular system by the various tests designed to gage its circulatory capacity. Other functions of the body, such as the metabolic rate, the sugar tolerance, etc., are determinable by the aid of microscopic, chemical, physical and specially designed instrumental tests.

Renal Function Tests

The methods of determining the function of the kidneys can be outlined in the following manner:

The Dye Tests

Of these tests the phenolsulfonephthalein and indigocarmine are best known. They depend upon the ability of the kidneys to excrete a given amount of a substance injected intramuscularly.

Technic: The patient is to drink 200 to 300 cc. of water, and in a half hour after that, 1 cc. of a solution containing 6 milligrams of dye substances is injected into the muscles of the back. At the end of an hour and ten minutes, the patient is to empty his bladder and the quantity of dye in the urine is then estimated by means of a colorimeter; this procedure is repeated at the end of two hours. Under normal circumstances in the two hours there is a return of 60 or 80 per cent of the dye. Less than this or, generally speaking, 50 per cent or less excretion is taken to mean kidney inefficiency. There are certain fallacies in this test which must be borne in mind. If the patient is edematous, the dye will not be readily absorbed, and, therefore, not readily excreted. If the patient has been purged severely, particularly with magnesium sulfate, the excretion of the dye will be interfered with, and again, although the method is simple, the technic of the test must be exact.

To test the efficiency of each kidney separately, phenolsulfonephthalein or indigocarmine is injected into the muscles of the back, both ureters are catheterized and the rapidity with which the dye appears from each kidney is noted

Nitrogenous End Products Determination

The determination of the retention of certain substances in the blood, such as urea, nonprotein nitrogen, uric acid and creatinin is probably the most valuable of all tests for determining the type of kidney lesion, and is of great prognostic significance (SEE. Blood Chemistry, p. 1007).

Urea Concentration Tests: These are made in order to determine the ratio between the blood urea and the urea in the urine during a given period. The best known of these tests is spoken of as Ambard's coefficient, but the method is so difficult technically, and its interpretation is fraught with such hazards that it is not generally useful and is rarely employed outside of large hospitals or research institutes.

Urea Concentration Test of MacLean and de Wesselow: The patient is given a definite amount of urea dissolved in water, one-hour samples of urine are collected, and the concentration of the urea determined.

Procedure: The patient is asked to empty the bladder, and immediately afterwards he receives by mouth 15 grams of urea dissolved in about 100 cc. of water. The bladder is emptied one hour and again two hours after the urea has been given and the specimens of urine are examined for the urea content. Thus, if urea is given at 10 A. M., a specimen of urine is obtained at 11 A. M. and at 12 NOON. The urea is then determined quantitatively If either specimen gives a percentage of urea above 2, the kidneys are held to be fairly efficient; the higher the concentration the more effective is the renal function. The reason why two specimens are taken is that in certain patients the urea given by mouth may produce a diuresis which tends to dilute the urine passed during the first hour. In such cases the second hour's specimen is important and should be examined. Indeed, in routine work it is generally best to discard the first specimen altogether, and to rely on the result obtained from the examination of the second specimen. Not more than about 120 cc. of urine should be passed in the second hour. Occasionally, if there is much available fluid in the patient's system, it may be necessary to take a specimen after three hours, or even to repeat the test In patients with marked diuresis this must be allowed for in estimating the renal function (Hawk and Bergeim).

Blood Urea Clearance Test: The urea of the blood and the amount excreted by the kidneys bear a close relationship in health. In disease of the kidneys, the urea clearance test will indicate kidney deficiency before excessive amounts of nonprotein nitrogen are discoverable in the blood. The normal rate of urea clearance is directly proportional to the blood urea concentration, and to the square root of urinary volume per unit of body weight. When the excretion rate of the urine is 2 cc per minute during the test period, it is assumed that about 75 cc. of blood is cleared of its urea content each minute during the same period. This is known as the "maximal clearance." When 1 cc. of urine is excreted per minute, which is known as the "Standard Clearance," only 54 cc. or about 40 per cent less is cleared during each minute The 2 cc rate of urine excretion and the 1 cc rate of urine excretion per minute are considered as standards, and the results obtained by examining the urea content of the blood after the patient has been properly prepared are expressed in terms of percentages of these standards. The urea clearance is considered normal when it is between 75 and 100 per cent of the standard.

Procedure: Two or three glasses of water are taken before breakfast; soon thereafter the bladder is completely emptied, and the time is noted This urine is discarded, and the test period begins

at this time. Breakfast is then taken. One hour after the beginning of the test period, urine is voided, the specimen is measured and saved, and at this time also 10 cc. of blood is taken from a vein and its urea content is noted. One hour later or two hours after the beginning of the test period, the bladder is again emptied completely; the two specimens of urine passed during the test period are measured and their urea content is noted. Comparison is then made between the urea concentration of the blood and of the urine.

Low urea clearance indicates impaired kidney function.

Mosenthal Test: This test depends upon the individual's capacity to concentrate his urine, as is determined by the quantity of urine excreted during the day and night. The specific gravity and quantity of urine passed every two hours during the day—8 A. M. to 8 P. M. is compared with the specific gravity and quantity passed during the night—8 P M. to 8 A. M. Under normal circumstances there should be a variation of at least nine points in the specific gravities of the two-hour specimens, and the total night urine should be less than 750 cc. (usually less than 450), and in the proportion of about one-half or one-third of the amount of day urine. The excretion of salt and of nitrogen should be at least one per cent. If the specific gravities vary less than nine points and if the night urine is large in amount and of low specific gravity, and the excretion of nitrogen or salt is insufficient, or if all or any one of these occur, it is taken as evidence that kidney efficiency is below par. This test is most useful as an aid to the early diagnosis of chronic nephritis, especially the type in which hypertension and nitrogen retention occur, that is, the type spoken of as chronic interstitial nephritis.

Fluid Concentration Capacity: When the kidneys are normal, the concentration of the urine under ordinary circumstances depends largely upon the quantity of water ingested. When small quantities of water are taken a concentrated urine of high specific gravity is voided, and when large quantities of water are taken, the urine passed is diluted and of low specific gravity. In advanced nephritis, presenting urine of a low specific gravity, the concentration power of the kidney will be found to be very low No matter how concentrated and dry the diet may be, the specific gravity of the urine will remain low. Also when large quantities of fluids are taken, they will have no effect upon further lowering the specific gravity of the urine.

In commenting upon renal tests in general, it is necessary to state that no one test is ideal, and that often all methods must be employed, as the kidneys react differently to the various bodies which are brought to them from the blood for excretion, and must, therefore, be judged separately in regard to their ability to excrete each one Like other laboratory tests they do not in themselves make a diagnosis, but are useful when added to the first hand clinical knowledge of the patient.

Liver Function Tests

The liver possesses a number of functions which play an important part in digestion and metabolism. Disease of the liver may be manifested by an interruption or perversion of one or several of its functions. By laboratory tests, several of the functions may be checked up. The functions of the liver, so far known, are:

1. Bile secreting function; 2. Glycogenic function; 3. Urea forming function (or destroying uric acid); 4. Detoxifying function; 5. Bactericidal function; 6. Lipogenic function; 7. Iron metabolism function; 8. Erythrocytic function.

Of all the known liver functions and possibly of many unknown functions that the liver possesses, only a few may be investigated by laboratory methods, *i. e.*, the bile secreting function, the glycogenic function, and possibly one of its digestive functions.

Bile Secreting Function

The quantity of bile absorbed in the circulation, either because of obstruction to the outflow of bile into the intestines or because of hemolysis, may be investigated by the following tests

Icterus Index (Bernheim): This is a method by which the quantitative amount of bile pigment in the blood serum is estimated colorimetrically. The normal icterus index is between 2 and 5; in clinical jaundice, the index may reach from 15 upwards Bile pigment in the blood in excess of the normal quantity may not be visibly recognized when its index is below 15 (the zone of latent jaundice range) Bilirubin is found normally in blood serum in proportions of *1 part to 500,000 When the bilirubin* content of the blood reaches to 1 part in 50,000, jaundice becomes visible. An icterus index from 10 to 20 may be seen in cholangitis, cholecystitis, cholelithiasis, hepatic cirrhosis, carcinoma and gumma of the liver, various inflammatory conditions of the liver and in adhesions of the gallbladder The icterus index may also be high in hemolytic jaundice, cardiac decompensation, internal hemorrhages and in fevers such as malaria, typhoid and pneumonia. This test is the most desirable for the quantitative estimation of bilirubinemia because of its simplicity, accuracy, definite clinical value and its safety. The icterus index only measures the quantity of bilirubin in the blood stream. Its clinical interpretation, however, depends upon the factors that produce this condition The icterus index test, to be of value, should be made at regular intervals in order to determine whether the jaundice is increasing, diminishing, or is stationary (SEE: p. 601).

The Van den Bergh Test: In this test the serum is treated with Ehrlich's diazo reagent which causes a red coloration when the bilirubin is present. The depth of color and the rate of its appearance is taken as an index of type and extent of bilirubinemia. Two types of reaction occur, *one, direct,* which may be (*a*) prompt reaction, (*b*) delayed or negative, reaction, or (*c*) biphasic reaction; and *two, indirect* (SEE: p. 602).

Direct Reactions: The three types of *direct Van den Bergh reaction* are said to be caused by chemical or physiochemical differences in the bilirubin and are attributed to the path by which the pigment enters the blood serum. *Prompt direct reaction* is seen in cases of frank obstructive jaundice; *delayed direct reaction* is seen in cases of hemolytic jaundice, *biphasic reaction* (two reactions are obtained, one prompt reaction and the other delayed reaction which is probably caused by the presence of two kinds of bilirubin in the serum) indicates that both obstruction and hemolysis are present in the same case This reaction often occurs in destruction of liver cells as in toxic or infective jaundice.

Serum yielding a direct Van den Bergh reaction indicates that the bili-

at this time. Breakfast is then taken. One hour after the beginning of the test period, urine is voided, the specimen is measured and saved, and at this time also 10 cc. of blood is taken from a vein and its urea content is noted. One hour later or two hours after the beginning of the test period, the bladder is again emptied completely; the two specimens of urine passed during the test period are measured and their urea content is noted. Comparison is then made between the urea concentration of the blood and of the urine.

Low urea clearance indicates impaired kidney function.

Mosenthal Test: This test depends upon the individual's capacity to concentrate his urine, as is determined by the quantity of urine excreted during the day and night. The specific gravity and quantity of urine passed every two hours during the day—8 A. M to 8 P. M is compared with the specific gravity and quantity passed during the night—8 P. M to 8 A. M. Under normal circumstances there should be a variation of at least nine points in the specific gravities of the two-hour specimens, and the total night urine should be less than 750 cc. (usually less than 450), and in the proportion of about one-half or one-third of the amount of day urine. The excretion of salt and of nitrogen should be at least one per cent. If the specific gravities vary less than nine points and if the night urine is large in amount and of low specific gravity, and the excretion of nitrogen or salt is insufficient, or if all or any one of these occur, it is taken as evidence that kidney efficiency is below par. This test is most useful as an aid to the early diagnosis of chronic nephritis, especially the type in which hypertension and nitrogen retention occur, that is, the type spoken of as chronic interstitial nephritis.

Fluid Concentration Capacity: When the kidneys are normal, the concentration of the urine under ordinary circumstances depends largely upon the quantity of water ingested. When small quantities of water are taken a concentrated urine of high specific gravity is voided, and when large quantities of water are taken, the urine passed is diluted and of low specific gravity. In advanced nephritis, presenting urine of a low specific gravity, the concentration power of the kidney will be found to be very low. No matter how concentrated and dry the diet may be, the specific gravity of the urine will remain low. Also when large quantities of fluids are taken, they will have no effect upon further lowering the specific gravity of the urine.

In commenting upon renal tests in general, it is necessary to state that no one test is ideal, and that often all methods must be employed, as the kidneys react differently to the various bodies which are brought to them from the blood for excretion, and must, therefore, be judged separately in regard to their ability to excrete each one. Like other laboratory tests they do not in themselves make a diagnosis, but are useful when added to the first hand clinical knowledge of the patient

Liver Function Tests

The liver possesses a number of functions which play an important part in digestion and metabolism Disease of the liver may be manifested by an interruption or perversion of one or several of its functions. By laboratory tests, several of the functions may be checked up. The functions of the liver, so far known, are:

Hippuric Acid Test

This test is based on the ability of the liver to synthesize glycine with benzoic acid and form hippuric acid which is eliminated in the urine.

Procedure: Six grams of sodium benzoate is administered, and the urine is collected during the following four hours. *In the normal,* approximately three grams of benzoic acid in the form of hippuric acid is excreted in the urine during the four hours after the administration of the sodium benzoate.

Diminished excretion of hippuric acid is found in hepatitis, portal and biliary cirrhosis, carcinoma, and syphilis of the liver and in hepatic necrosis, also in catarrhal jaundice and in chronic hepato cellular degeneration.

Normal finding occurs in jaundice due to uncomplicated obstruction of the common bile duct and in gallstones. This test is therefore of value in differentiating between jaundice due to uncomplicated biliary obstruction and hepato cellular disease.

Takata-Ara Reaction

A positive Takata-Ara reaction is obtained in the presence of a high globulin content of the serum, especially when the albumin fraction is decreased. This test is also positive in a large proportion of cases of portal cirrhosis. It is, however, recognized that a positive reaction is the result of a high globulin content of the serum and is not a specific test for liver damage.

Cholesterol Content of the Blood

In certain diseases of the liver, the cholesterol content of the blood may be increased or diminished. When the cholesterol esters (combined with fatty acids) fall below 50 per cent of the total cholesterol content of the blood in hepatic disease and particularly in common duct obstruction, it is an indication of liver damage and is of serious prognostic significance. *Hypercholesterolemia* is also found in myxedema and milder forms of hypothyroidism. *Hypocholesteremia* is found in hyperthyroidism and exophthalmic goiter.

The Cephalin-Cholesterol Flocculation Test: This test was introduced by Hangar as a means of differentiating obstructive from hepatogenous jaundice. In actual liver damage, the normal 2:1 albumin-globulin blood serum ratio is greatly reduced or may even be reversed because as the quantity of albumin in the serum decreases, the globulin may remain unchanged or increased. The pathologic globulins, when brought in contact with a cephalin-cholesterol emulsion, will cause various degrees of flocculation, depending upon the amount of hepatitis.

Procedure: 0.2 cc. of patient's fresh serum is mixed with 4 cc. of normal saline in a test tube; to this is added 1 cc of properly prepared cephalin-cholesterol emulsion, and the contents are thoroughly mixed. The tube and its contents are allowed to stand in the refrigerator and are examined at the end of 24 and 48 hours for the presence or degree of flocculation. (A control tube containing 4 cc. of saline solution and 1 cc of emulsion without serum should be placed in the same refrigerator for comparison and for testing the stability of the serum.) With normal sera, the emulsion remains as a stable homogeneous suspension; but with sera from patients with liver damage, the emulsion

rubin contained in the serum has passed through the liver cells. This is found in biliary obstruction and in hepato cellular disease.

Indirect Reaction: The bilirubin content of the normal blood has been found to be 1 in 1,800,000 to 1 in 500,000. Van den Bergh takes 1 in 200,000 as a unit. The limits are 0.1 to 0.5 units. The renal threshold value of bilirubin is approximately four units, because bile *does not appear in the urine until four units are present in the blood. In hemolytic jaundice, this relation does not* hold, as it is possible to have between 5 and 18 units in the blood with no bile in the urine. This is possible because the bile *may be excreted in the form of* urobilin. Latent icterus is a condition in which there is sufficient bile to produce slight jaundice but no bile appears in the urine.

Blood which fails to *yield* a positive *direct reaction may on the addition of 95 per cent alcohol yield a violet color; that indicates an indirect reaction.* Serum yielding only an indirect reaction indicates that its bilirubin content has not passed through the liver cells. This reaction is found in hemolytic jaundice, pernicious anemia, erythroblastosis, sickle cell anemia, in absorption of blood from the peritoneal cavity, and in newborn babies.

Bile Test: Liver function is also studied by chemical and microscopic examination of the bile. *The bile may be obtained direct from the duodenum by* Lyons' method of biliary drainage, and is studied microscopically for various bacteria, crystals, inorganic salts, bile pigments and liver cells. The amount and quantity of bile secreted by the liver can also be determined by duodenal *drainage. The rate at which the bile flows through the tube is often an indication of the rapidity of bile secretion;* a very slow *flow of bile may indicate* partial obstruction of the gallbladder or bile ducts.

The bile obtained by drainage is classified by Meltzer and Lyons as follows: "A" bile.—The contents of the duodenum and common duct are a yellowish green alkaline fluid, the first to appear through the drainage tube. *"B" bile.—The contents of the gallbladder are viscid, concentrated and darker, the second* portion of bile. "C" bile.—The contents of the hepatic ducts are watery and lemon yellow or greenish in color, the *third portion of bile.*

If "A," "B," "C" bile is secured through the tube, it may be assumed that the gallbladder is functioning properly. If "A" and "B" bile are found to contain clumps of cholesterin crystals, *gallstones in the common duct and gallbladder may be suspected. If the "B" bile alone contains clumps of cholesterin* crystals, cholelithiasis may be suspected. The absence of cholesterin crystals and the presence of bile-stained epithelial débris and bacteria indicate cholecystitis.

Serum Phosphatase: The normal serum phosphatase in adults is 1 5 to 4 Bodansky units (0.10 to 0.21 Kay units), and in growing children 5 to 14 Bodansky units.

The serum phosphatase is increased in *obstructive* jaundice, hepato cellular jaundice, portal cirrhosis, carcinoma of the liver, biliary fistula and in osteitis fibrosa cystica, osteogenic sarcoma and other destructive bone diseases.

Normal values are obtained in *hemolytic* jaundice and congenital *atresia of* the bile ducts.

venously and estimated liver function by the amount of dye eliminated in the stools.

The Glycogenic Function

Levulose Test (Sachs-Strauss): One hundred grams of levulose are given by mouth. The appearance of sugar in the following 24 hours' specimen of urine is considered to be an indication of liver dysfunction. Spence and Brett considered the urinary findings of this test unreliable because they found that the kidney threshold for levulose is lower than for glucose and that it varies greatly in individuals. They administer 50 grams of levulose by mouth and examine the blood for sugar. A rise in blood sugar indicates liver insufficiency The degree of insufficiency may be judged by the height of the blood sugar curve. The Sachs-Strauss test was positive in most of their cases of early jaundice except of the hemolytic variety

Widal Hemoclastic Crisis Test

When 200 cc of milk is given to a normal person, the blood will show a distinct digestive leukocytosis The absence of leukocytosis is taken by Widal to be an indication of liver dysfunction. This test is considered by many clinicians as unreliable

The Indican Reaction

The liver is supposed to destroy indican, hence spontaneous indicanuria, or indicanuria after a provocative dose of indol, is said to indicate liver inefficiency Tests depending upon a increase in the ethereal sulfates after the administration of substances such as thymol have been described, but since these reactions have never been in general use and have proved unsatisfactory, they will not be discussed.

Basal Metabolism

Metabolism may be defined as the process of building up and breaking down of tissues within the body. The rate at which this process is going on in the body differs with the age, sex and size of the individual. Certain diseases are characterized by an increased metabolic rate, *e. g.*, hyperthyroidism, while other diseases are characterized by a low metabolic rate, *e. g.*, hypothyroidism and hypopituitarism.

Basal metabolism is an expression of heat production, oxygen consumption and carbon dioxide elimination of the body while completely at rest and not under the influence of stimulating food, or excessive variations in the temperature and barometric pressures. In testing for basal metabolism, one of several makes of apparatus may be chosen for use. The Sanborn graphic apparatus, because of its simplicity in operation and compactness, is a good instrument for office or bedside use, though any standard make can be employed with accuracy

Technic and Interpretation: Preliminary to the test, the patient shall not take food or liquor for 14 to 16 hours in order to avoid the effect of stimulation caused by exogenous metabolism and glandular secretion. Perfect rest and relaxation of at least one or two hours are necessary before the test is begun. During the test the patient is to lie thoroughly relaxed with all tight lacings removed, and should not be permitted to read. Excitement and emotions of any kind are to be avoided. After having the patient in the proper position and the apparatus previously tested, it is connected to the patient, who breathes regularly, thus inspiring a quantity of oxygen over a definitely stated period.

The amount of oxygen consumed by the patient as indicated by the apparatus

tends to flocculate and to precipitate to the bottom of the tube, leaving a clear solution. The readings of the solution are recorded as 0, ±, +, ++; complete flocculation is recorded as +++, ++++; a clear solution as 4+. False positives may occur when the cephalin-cholesterol emulsion is not properly prepared A negative flocculation reaction in one who has previously shown a positive reaction, notwithstanding the persistence of jaundice, is a favorable omen, indicating liver repair On the other hand, a persistent strongly positive flocculation reaction, irrespective of the degree of jaundice, is of grave prognostic import, as it usually indicates continuous liver damage.

Prothrombin Time and Vitamin K as a Liver Function Test: Among the many functions the liver possesses is the formation of plasma prothrombin To accomplish this, vitamin K is essential In order to utilize vitamin K from the food for the formation of prothrombin, there must be present a sufficient amount of bile in the gastrointestinal tract Prothrombin formation may be interfered with either by disease of the liver or by vitamin K deficiency. To determine the cause of jaundice, whether extrahepatic (obstructive) or intrahepatic (cellular), the vitamin K test may be of value.

Technic and Interpretation: A specimen of blood is drawn for the determination of the plasma prothrombin level, and at the same time 2 mgm. of vitamin K is injected intramuscularly. At the end of 24 hours, another specimen of blood is drawn and the prothrombin level is determined. A rise of the serum prothrombin of 10 per cent or more is an indication of extrahepatic jaundice. If a rise of less than 10 per cent is noted in the second specimen, a third is taken at 48 hours or a fourth at 72 hours. If a rise of 15 per cent in either of these specimens over the first is found, then the jaundice may still be considered as being extrahepatic If, however, there is no rise or a rise much lower than 15 per cent above the initial level, the jaundice may then be considered as being of intrahepatic origin; that is, due to liver damage

The Dye Tests

Dye in the Blood Serum: Bromsulfalein Test (phenoltetrabromphthalein sodium sulfonate Rosenthal and White): This test, as an indicator of hepatic function, depends upon the rapidity with which the dye is removed from the serum. Normally, the intravenous injection of two milligrams of the dye per kilogram of body weight is completely removed from the blood in 30 minutes; in liver disease, the dye may be retained in the blood in various concentrations up to 100 per cent of the amount injected The percentage of the dye present in the serum half an hour after injection indicates the degree of liver function impairment

Dye in Bile: Phenoltetrachlorphthalein Test (Aaron, Beck, Schneider, Piersol and Bockus): The dye is injected intravenously in order to determine the ability of the liver to excrete it. When the liver is normal, the dye, after intravenous injection, can be detected in the bile obtained by the duodenal tube in from 12 to 17 minutes. In various liver diseases, the appearance of the dye in the bile is very much delayed.

Rowntree employed this dye intra-

a current produces a magnetic field which acts at right angles to its course, varying according to the intensity of the current, and proportionately attracting or repelling another adjacent magnetic field.

The electrocardiograph is but an improved and modernized string or beam galvanometer in which the heart's action is recorded by projecting its shadow, magnified by a high power microscope, on a camera mechanism The time is recorded by the shadow of a rotating spoked wheel activated by a tuning fork

In order to depict the curve of the heart's action, the instrument is brought into contact with the patient's body by electrodes; these are connected at four or more different planes known as *Leads*.

Lead I Two electrodes are connected one to each forearm, and the curve thus obtained is derived largely from the base of the heart, and is also known as derivation 1

Lead II The electrodes are connected one to the right arm and the other to the left leg. This corresponds to the long axis of the heart, and is known as derivation 2

Lead III The electrodes are connected to the left arm and to the left leg, corresponding largely to the left side of the heart, and is known as derivation 3.

Lead CF-2 The left leg electrode is applied in the third intercostal space at the left sternal border; the right arm electrode is applied to the left leg

Lead CF-4 The chest electrode is shifted to the fourth intercostal space at the left midclavicular line; the left leg electrode remains as in CF-2

Lead CF-5 The chest electrode is placed in the fifth intercostal space at the left anterior axillary line, the left leg electrode remains as in CF-2 and CF-4

Other chest leads are taken by shifting the chest lead to the left midaxillary, posterior axillary, and midspinal lines. CF-4 is the most important of the chest leads. When only one chest lead is taken, it should be at the CF-4 level.

When clinical symptoms suggest myocardial infarction which is not revealed by leads I, II, III, and CF-4, other chest leads should be taken.

Most of the electrocardiographs possess a special electrode for lead IV; this obviates the necessity of changing the electrodes from the arm and leg. Only the lead IV electrode is shifted from one point upon the chest to another in order to get each required CF lead.

The first three leads are known as the indirect leads, the others as the chest or direct leads.

The contracting heart constitutes an area of electropotentiality and the derivations may be regarded as planes which transect this electric field. Electrocardiography may, therefore, be defined as the study of the direction, amplitude and time of the cardiac action currents graphically expressed (Willius). It is a graphic curve depicting the path of the heart wave as it progresses along the conduction system of the heart, from the sinoauricular node of the auricle to its final distribution. The origin and distribution of the waves, either normal or abnormal, are recorded upon bromide paper by the electrocardiograph The camera mechanism consists of an absolutely dark box and a moving film which is controlled either by the release of an oil cylinder or by a motor rotation arrangement. The speed of the moving film can be regulated accurately. The coarse and fine ordinate markings of the electrocardiograms indicating fifths and twenty-fifths of a second result from the shadow of the rotating spoked wheel.

Basal Metabolic Rate
Normal Values From +15 to −15

The basal metabolic rate is *increased in*:	
Exophthalmic goiter, the metabolic rate bears a definite proportion to the severity of the disease and may range from	+20 to +150
Leukemia	+20 to +120
Cardiorenal disease with dyspnea and anxiety	+15 to + 50
Pulmonary tuberculosis *with high fever*	*+15 to + 40*
Pulmonary tuberculosis without fever and with thyroid enlargement	+10 to + 20
Diabetes mellitus (severe) with glandular enlargement	+ 5 to + 25
Nephritis without edema	+ 2 to + 30
Pernicious anemia	+ 2 to + 33
Typhoid fever	+15 to + 50
The basal metabolic rate is decreased in:	
Cretinism and myxedema	*−15 to − 60 or lower*
Hypopituitarism	−10 to − 40
Diabetes mellitus (severe) without glandular enlargement	− 1 to − 20
Diabetes mellitus (fasting)	−15 to − 36
Diabetes mellitus (with emaciation)	−10 to − 37
Nephrosis (Epstein's)	−10 to − 40
Nephritis with edema	+10 to − 40
Starvation	−10 to − 30
Pulmonary tuberculosis (afebrile)	+15 to − 33
Obesity	+10 to − 12

is the main factor in establishing the basal metabolic rate If the patient consumes a large amount of oxygen, this indicates a rapid combustion and means a high metabolic rate. If the amount of oxygen consumed is small, it indicates low combustion and is therefore evidence of a low metabolic rate.

The basal metabolic rate is determined by considering the amount of oxygen inhaled in a definite period and is computed from figures in a specially prepared table on "basal metabolism," which takes into consideration the age, stature, sex and weight of the patient, the room temperature and the barometric pressure. Normal metabolic values are considered from plus 15 to minus 15 Anything above plus 15 may be considered increased basal metabolism, while below minus 15 may be considered decreased basal metabolism The basal metabolic rate is usually greater in children than it is in adults, and in adults it is greater in the small, thin individuals than in the large, fat individuals. The basal metabolic rate is the best index by which thyroid disease may be judged.

Specific Dynamic Action of Proteins

The B. M. R of the patient is estimated in the usual manner. Then the patient is allowed the whites of three boiled eggs, a slice of bread without butter and a cup of tea without sugar. After two hours of rest, the B. M R is again estimated Normally, the second B.M.R. shows an elevation of 15 to 18 per cent over the first one. In pituitary deficiency the elevation does not rise above five per cent

Electrocardiography

Electrocardiography is the study of the heart's action by depicting the electric waves created by the heart, which are transmitted by way of the extremities and the chest wall through the electrocardiograph upon an *electrocardiogram.*

The Electrocardiograph

The electrocardiograph is actually a galvanometer adapted for clinical purposes. The galvanometer works in accordance with the physical principle that

action and prolonged in slow heart action. Abnormally prolonged P-R interval occurs in A-V bundle block.

It is followed by a narrow, tall steeple-like spike called the *R wave,* which represents the beginning of the ventricular contraction. (The height of the R wave corresponds to the first sound of the heart.) This in turn is followed by a third wave, the *T wave,* which is higher than the P wave, but only one-third as high as the R wave; it represents the final activity of the ventricles.

Two other waves known as the Q-S waves are sometimes found at the base of the R wave. The Q wave is found at the right extremity, and the S wave is found at the left extremity The distance between the Q and S waves is significant. The deflections of the Q and S waves are represented by rather short, abrupt peaks directed downward; they blend with the ascending and descending limbs of the R wave.

Normally, the sequence P-R-T waves will occur for as long a period as one may choose to have the electrocardiograph in operation The extent of the electrocardiogram represents multiplications of the original P-R-T-Q-S waves, and the same should be observed in all three Leads Deviation from the normal P-R-T waves indicates a pathological condition. The letters P-R-T-Q-S, etc, have no particular significance except that they have been adopted to represent these waves by the early workers with the electrocardiograph, who employed these letters instead of the over-used first letters of the alphabet

The normal electrocardiogram consists of a series of waves or deflections which have been arbitrarily termed P-Q-R-S-T and U. The deflections are grouped according to their occurrence in the cardiac cycle; thus P is known as the auricular complex, and Q-R-S and T as the ventricular complexes. The deflections Q-S are important in the diagnosis of myocardial defects due to coronary occlusion and to other defects.

The amplitude of the R wave varies between 10 and 15 millowatts. The width of this wave normally does not exceed 0.10 of a second Because of the extremely rapid deflection of the galvanometer the R wave appears on the electrocardiogram as a delicate line

The Q-R-S complex lasts from 0 08 to 0 1 of a second.

The amplitude of the T wave is from three to seven millowatts and has a duration of about 0 27 of a second. The S-T interval is the distance between the S wave or the termination of the descending limb of the R wave when the former is absent and the end of the T wave, it has been shown by Mekens not to exceed 0 28 of a second (Willius) The Q-R-S-T complex represents the systole of the ventricles Its duration from the beginning of the Q or the base of the left limb of the R to the end of the T varies with the rate of the heart; it is usually between 0 32 and 0 42 of a second

Limitations of Electrocardiography: Electrocardiography can supply information concerning only the conduction system of the heart. It cannot give any information on diseased conditions of the heart valves, of the pericardium, or the endocardium, or of the aorta, or of the blood supply of the heart, unless —because of disease of these structures— the heart muscle becomes secondarily affected, thus interfering with the conduction path of the heart's impulse It is inadvisable to base a diagnosis on the

produced by etched lines in the camera lens. In order to appreciate the normal and abnormal waves depicted upon an electrocardiogram, it is necessary for the paper to revolve at a given velocity during the recording process. There are also arbitrary divisions by which the height and length of certain waves may be measured. These divisions are formed into squares by delicate cross lines, each large square being 0 2 of a second wide and five millimeters high.

Three primary waves represent the contraction of the heart; the *P wave* is seen on the electrocardiogram as a small *rounded elevation; normally, it is always* directed upward. It has a deflection (amplitude) of from two to four millowatts (milliolots), *its duration is 0 1* second, and is closely followed by the Q-R-S complex. The P-R *wave interval, the distance from the beginning of* the P wave to the beginning of the R wave, normally occupies 0 14 *to* 0 20 of a second; this represents the contraction of the auricle. The A-V node is reached at about the summit of the P wave. The P-R interval is shorter in rapid heart

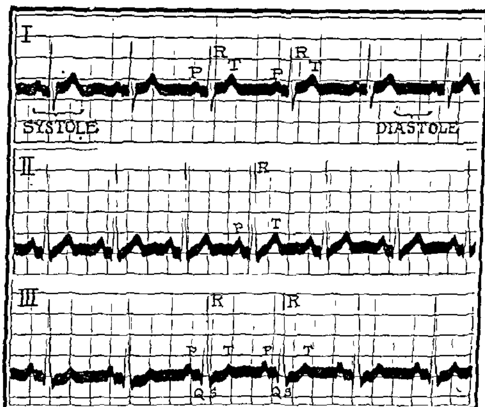


Fig 1—Normal electrocardiogram A complete clinical electrocardiogram consists of three records, called *Leads* The first is taken across the base of the heart the second along the right

1. The auricular and ventricular rates are compared by counting the P waves and the R waves which occur in 20 squares of the record (four seconds) and multiplying by 15 to get the number of beats per minute.

2. The origin of the heart rhythm is to be determined, *i e.*, whether it originates in the sinoauricular node, as in the normal, or at some abnormal point.

3. The conduction time from auricle to ventricle is estimated from the beginning of the P wave to the beginning of the R wave. When the P-R interval is greater than 0.2 of a second, it indicates delayed conduction; such a delay is noted in heart block.

4. Departures from the normal waves in any leads should be noted, as it is thus possible to tell in which chamber of the heart the abnormality is located.

The Polygraph

The polygraph is an instrument devised to take the tracings simultaneously of an artery (arteriogram), of a vein (phlebogram), and of the cardiac apex beat (cardiogram).

It consists of a recording apparatus, which has two or three airtight rubber tubes attached to it. The ends of the tubes are fitted with cups, one adjusted to the jugular bulb or liver; the other to the radial artery, and a third over the area of the apex beat. The other ends of the tubes are so connected to the recording apparatus that the pulsations perceived by the cup and transmitted along the tube cause an inked pen to oscillate These oscillations are recorded upon a strip of paper which is being revolved by a clock mechanism, the speed of which can be regulated The tracings upon that paper constitute a *polygram.* Clinically the electrocardiograph has largely supplanted the polygraph, but has not entirely displaced it.

Phlebograms: A *phlebogram* may be recorded either from the jugular bulb or from a pulsating liver. When this is compared with the tracings of an arteriogram (sphygmogram) it enables one to estimate the conduction time from auricle to ventricle.

The three principal waves of a phlebogram are the A-C-V waves The A wave is thought to be due to auricular systole, and represents auricular contraction The C wave represents ventricular contraction. The interval from where the A wave begins to where the C wave commences in the jugular tracing is said to represent the conduction time from auricle to ventricle, and is known as the A-C conduction-time interval The V wave is caused by an increased pressure in the veins, which is probably due to regurgitation of blood in the veins, and the rise of pressure in the auricles because of ventricular systole. The A-C and V waves are positive waves. There are also three negative waves, the X-Y-W waves, these are caused by the negative phases in the circulation, when pressure is suddenly removed from the veins

Heart block can be recognized by a multiplicity of A waves, and auricular fibrillation by the absence of recurring A waves The source of premature contractions may be identified by noting a premature A or C wave

Arteriograms (Sphygmogram and Cardiogram): An *arteriogram* is obtained from any superficial pulse or from the cardiac impulse. Usually the radial artery is chosen for this purpose. The sharp upward wave of an arteriogram is termed a *percussion wave.* This is fol

data obtained by a cardiographic examination alone, because an instrument so fine as the electrocardiograph may occasionally *produce erroneous data*, and because the condition of the patient's heart action as reproduced on the electrocardiogram represents only what is going *on during the brief time required for* the examination The electrocardiograph is still a comparatively new addition to the clinical armamentarium. Electrocardiography has a definite place in medicine, but it should by no means be permitted to displace a thorough physical examination. For Electrocardiographic Interpretation of Heart Action, SEE · p 435.

Definitions of Terms Used in Electrocardiography

Wave is an elevation produced by the *contraction of the auricles or ventricles; for instance, P-R-T waves*, etc

Leads are records obtained from a single source We speak of four leads: I. The arm lead; II. The right arm and the *left* leg lead; III. The left arm and left leg lead; IV. The left chest and left leg lead. The four leads are records which form a complete clinical electrocardiogram.

Waves are divided into the ascending limb, the upstroke of the wave; the summit or plateau, the *uppermost* portion of the wave; and the descending limb, downward stroke of the wave

Positive refers to a wave when directed upward; negative *refers to* a wave when directed downward.

Amplitude and ***voltage*** are terms used to express the excursions of the waves. The term "low amplitude" is applied to a lowering or flattening of a single wave as in a flat T, indicating *pathology in that part of the heart which* is responsible for the production of the wave Low voltage designates low amplitude in all waves and in all leads. It indicates either a diminished *production* of electricity within the heart or interference of the heart's current in reaching the extremities.

Isoelectric refers to a flat wave; diphasic, when the wave starts in one direction, then sharply slants in another.

Slurred is used *when either the* ascending or the descending limb of a wave is heavier than the rest of the stroke

Notching is a sharp depression or a notch in part of the wave.

Splintering signifies multiple notchings of a wave

Tremors are fine elevations as a result of vibration of the base line obtained *from graphic records of nervous, emotional people who are under muscular* tension Tremors may be of somatic origin, when due to vibration of skeletal muscles; visceral tremors are caused by *visceral muscles.*

Emming (M-ing) or ***double-uing*** (W-ing) signifies the splintering of a wave to resemble the letters *M or W*. This is found chiefly in the ascending or descending limbs of the R wave

Analysis of Records

In the interpretation of heart records, all four or more leads should be considered. *It is advisable for the beginner in* this kind of work to keep a normal tracing before him with which to compare the abnormal curves he desires *to interpret.*

The information to be sought from the study of an electrocardiogram is as follows:

"(*f*) Bigeminy is most often due to premature ventricular contractions.

"(*g*) To differentiate bigeminy and alternation: Alternation is always late or evenly spaced—never premature; bigeminy, however, is premature.

"(*h*) When a run of regular beats occurs in a grossly irregular polygraphic tracing, think of auricular flutter, but confirm the thought by electrocardiography."

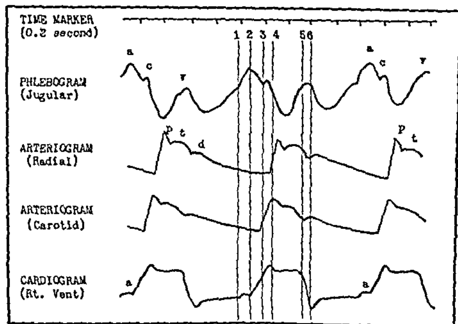


Fig. 3—The time of events in waves of the polygram. The perpendicular lines represent the following events (1) Auricular systole begins, (2) ventricular systole begins; (3) the pulse appears in the carotid; (4) the pulse appears in the radial; (5) the semilunar valves close, (6) the tricuspid valve opens (adapted from Hay). (*S. Calvin Smith*, F. A. Davis Company)

The Sphygmograph

The *sphygmograph* is an instrument for registering the movements, form and force of the arterial pulse. The general principle of the instrument is as follows: A steel spring is laid upon the radial artery at the wrist, so that it partially compresses the artery, and is moved up and down by the arterial impulse. Attached directly to the spring are a series of small levers which magnify the movement of the spring. The free extremity of the lever presses lightly against a strip of paper that has been blackened with the smoke of burned camphor or turpentine. This strip of paper by a clock arrangement moves at a uniform speed. When the tracing of the pulse is completed, the paper is preserved by submerging it in compound tincture of benzoin which covers it with a glaze (sphygmogram). When dried, it may be preserved as a permanent record.

Cardiac Function Tests. SEE: p. 442.

Peripheral Circulation Function Test. SEE: p. 543.

lowed by a second wave named the *tidal wave*, which terminates in a third wave known as the *diastolic notch*. The latter indicates the closure of the aortic valves and marks the termination of the pulse of the sphygmic period

The *cardiographic tracing* is obtained by applying the receiving cup to the apex beat, and shows graphically the strength of the ventricular systole, and the length of time in which the heart remains in

S. *Calvin Smith* gives the following suggestions for analyzing polygrams (for more detailed information, the reader is referred to S Calvin Smith's book on *Heart Records*).

"(*a*) The A wave is absent in any weak auricular action—as in auricular flutter or auricular standstill.

"(*b*) Expect to find a split-A in a heart block.

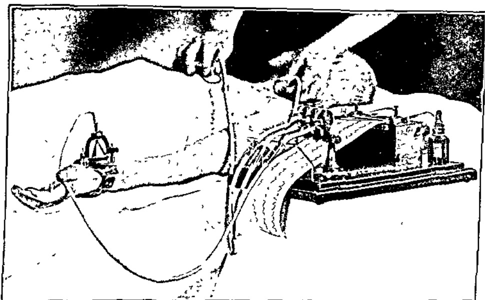


FIG. 2—The polygraph applied The position of the patient, receiving cups, and cushion tambour are shown above (*S. Calvin Smith*, F A. Davis Company)

contact with the anterior chest wall, and the period when relaxation of the heart begins. To be of value, a phlebogram should be compared with an arteriogram taken at the same time The arteriogram alone, however, may disclose sinus arrhythmia, pulsus alternans, premature contraction, heart block, and auricular fibrillation, but never auricular flutter However, when polygraphic studies are made, it is best to compare the phlebogram with the arteriogram and cardiogram.

"(*c*) Sometimes an A wave may be seen in the radial tracing of heart block; it is due to the impact of a dilated auricle on the aorta

"(*d*) A heart block is called complete when the a-c interval varies disproportionately in length—as 0 2 then 0 3, then 0 25 of a second, etc.

"(*e*) Any wave that persistently goes below the base line of the radial tracing is a deep dicrotic notch, and the following wave is a part of the preceding contraction, despite its deceptive height.

"(*f*) Bigeminy is most often due to premature ventricular contractions.

"(*g*) To differentiate bigeminy and alternation: Alternation is always late or evenly spaced—never premature; bigeminy, however, is premature.

"(*h*) When a run of regular beats occurs in a grossly irregular polygraphic

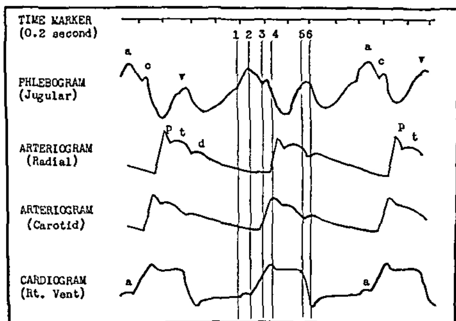


Fig. ...
followi...
pulse a...
close; ...
Company)

tracing, think of auricular flutter, but confirm the thought by electrocardiography."

The Sphygmograph

The *sphygmograph* is an instrument for registering the movements, form and force of the arterial pulse. The general principle of the instrument is as follows A steel spring is laid upon the radial artery at the wrist, so that it partially *compresses the artery, and is moved up and down by the arterial impulse.* Attached directly to the spring are a series of small levers which magnify the movement of the spring. The free extremity of the lever presses lightly against a strip of paper that has been blackened with the smoke of burned camphor or turpentine. This strip of paper by a clock arrangement moves at a uniform speed. When the tracing of the pulse is completed, the paper is preserved by submerging it in compound tincture of benzoin which covers it with a glaze (sphygmogram) When dried, it may be preserved as a permanent record.

Cardiac Function Tests. SEE: p 442.

Peripheral Circulation Function Test. *SEE: p. 543.*

CHAPTER XXXVII

Other Diagnostic Tests

Bacterial Identification

The various microörganisms are identifiable by (1) their manner of growth upon specific media, (2) their general morphology when properly stained, and (3) by their ability to reproduce the disease when inoculated in a nonimmune subject. Guinea pigs, rats, mice, or other laboratory animals are employed as culture media when growths of organisms are otherwise not obtainable.

Cultures may be taken from infected foci, wounds, mucous membranes, secretions, excretions and from the blood

Staining Affinities: A large number of microorganisms are stainable with methylene blue; their shape, size and characteristic formations are thus revealed under the microscope (oil immersion lens). Other organisms require special stains, counterstains and special staining methods, thus, the tubercle bacilli, the diphtheria bacilli, treponema pallidum, the spirochetes, etc.

There are also a number of organisms that easily resemble one another and may be identified only by their ability to change color when counterstained by the Gram's stain These organisms are spoken of as either Gram positive or Gram negative; few are also spoken of as Gram ambophile.

Gram's Method: Organisms are stained with glycerin crystal violet, or gentian violet for three minutes and then with Gram's iodine solution for one or two minutes, and washed in water and decolorized with 95 per cent alcohol, and again washed with water and counterstained with Bismark brown or safranin, or fuchsin. Gram-positive organisms are violet in color (They retain their violet color and do not take the counterstain)

Gram-negative organisms are decolorized with the alcohol and therefore assume the color of the counterstain

Gram ambophile organisms may be gram positive or gram negative

Gram-positive organisms (those that retain their original stain) are the following cocci. Pneumonococci, staphylococci (aureus and albus), streptococci, micrococci tetrageni; and the following bacilli, *i. e.*, the tubercle bacilli and other acid-fast bacilli, the diphtheria bacilli, bacilli subtilis, anthrax bacilli, tetanus bacilli, botulini, Welch's bacilli and other spore-bearing anerobes

Gram-negative organisms (those that take counterstain) are the following cocci: Meningococci; gonococci, micrococci catarrhalis, and the following bacilli· Typhoid; paratyphoid, dysentery, influenza, pertussis, Friedlander's, proteus, malleomyces mallei, pyocyaneus, tularensis, pestis, pusiformis, brucellae melitensis and others.

Gram ambophile are yeasts and mold, protozoa and older forms of gram-positive organisms

The Neufeld Method for Typing Pneumococci

Thirty or more different strains of pneumococci have been isolated; each strain gives a specific reaction (swelling of its capsule) when brought in contact with its hemologous type serum.

Technic: Several drops of typing serum faintly colored with alkaline methylene blue are placed on a slide to which is added a loopful of suspected sputum or a culture containing pneumococci This is covered with a cover slip and examined, preferably as a hanging drop, under an oil immersion lens with light partially dimmed. When the testing serum and pneumococci in the sputum are of the same strain, it will be noted that the capsules surrounding the pneumococci become greatly swollen in from 5 to 30 minutes. In order to isolate the proper type, the 30 known types of sera are to be tested against the sputum until the type is identified. When the number of pneumococci in the sputum are scarce, a droplet of sputum may then be injected intraperitoneally into a mouse Within 24 hours, the mouse may be killed and if found to have grown the pneumococci, these are then tested by the Neufeld method for proper identification.

Pregnancy Tests and Their *Clinical Values*

It often becomes necessary to determine the presence of pregnancy long before it is recognizable by physical examination Frequently neoplasms, pseudocyesis, or certain toxemias may simulate pregnancy, or a pyosalpinx may resemble tubal pregnancy To differentiate these conditions from pregnancy, certain biologic tests may be performed which will, in most cases, disclose the presence of pregnancy. The majority of pregnancy tests are based upon the great increase of estrus-producing hormone and of the anterior pituitary gonadotropic hormone in the blood and the urine of pregnant women, so that when a small amount of blood or of urine is injected into an immature or a virgin animal, definite estrus or maturation of ovarian follicles is produced.

The Aschheim-Zondek Test: The Aschheim-Zondek test as modified by S Aschheim is as follows: Five infantile mice are used for each test. The animals are weighed at the beginning of the experiment; they should weigh from 6 Gm. to 8 Gm. *unless they belong to* smaller or larger types. It is important that they be three to four weeks old and show no spontaneous sexual maturation The first urine passed in the morning is injected into the animals subcutaneously, in six doses. Six doses of 0 5 cc. are injected into each of five animals, three doses on the first day and three on the second day. (Many urines are quite toxic Some of these toxic urines, but not all of them, may be detoxicated by shaking them up with ether in accordance with the method proposed by Zondek.) On the fourth and fifth days, vaginal smears are made; 96 hours after the beginning of the test the animals are killed and the ovaries examined for corpora lutea and blutpunkte. These may usually be seen with the naked eye but more readily with a lens. Microscopic examination of the ovaries is seldom necessary. Such examinations are made only to establish the occurrence of reaction I in case a positive Allen-Doisy test shows a definite hormone effect but the corpora lutea and hemorrhagic spots are not apparent. In such an event the urine is subjected to a second examination.

Positive results sometimes may be obtained as early as 60 hours after the first injection. In this case it is advisable to use several more animals and to kill only half of them at 60 hours. If a positive diagnosis is not made at this time, the result is checked with the remaining

animals at 96 hours. In emergency cases the Friedman method, which employs mature rabbits, may be used; with this method diagnoses may be made in 24 hours.

The Friedman Modification of the Aschheim-Zondek Test: The Friedman modification is now used more extensively because (1) rabbits are easier to obtain, (2) the diagnosis can be made 36 hours after the test instead of waiting 96 hours, (3) no microscopical examinations are necessary—the "reaction" is ascertained from the gross appearance of the gonads. This test depends upon an excess of the maturation hormone in the urine during pregnancy, and also upon the fact that female rabbits ovulate normally only after mating, but after intravenous injections of urine containing an excess of the maturation hormone, the ovaries of the rabbit respond in 24 to 48 hours by the formation of corpora hemorrhagica and lutea.

Friedman uses rabbits because, although the ova in the rabbit ripen continuously, ovulation does not occur until after copulation. He was therefore able to study the effect of the urine of a pregnant woman (due to the presence of a pituitary-like hormone in the urine) on the ovaries of such rabbits free from corpora hemorrhagica and corpora lutea.

Procedure: Ten cc. of clear urine is injected slowly into an ear vein of a female rabbit about four months old and weighing about four pounds. The rabbit must have been in isolation for a month or her ovaries examined by laparotomy prior to the experiment. Twelve hours later another injection of 10 cc. of clear urine is made. Twenty-four hours after the second injection, the rabbit is killed and autopsied immediately. A positive reaction is indicated by subserous hemorrhagic areas and, sometimes, corpora lutea.

Significance of the Test:

1. Living fetus or placenta
2. Hydatidiform mole.
3. Chorionepithelioma.
4. Malignant tumor of testes (seminoma).

The Mazer-Hoffman Test (Estrin Test): This test is based upon the changes produced in the vaginal mucous membrane of a castrated adult female mouse after the injection of 15 cc. of whole urine in six divided doses over a period of two days. The reaction is considered as positive when, after the third day, there appears in the vaginal smear "a preponderance of nonnucleated epithelial cells and the absence of leukocytes and mucus." From the first to the eighth week of pregnancy, one liter of urine is supposed to contain from 300 to 600 mouse units of estrin; that is, four mouse units to 15 cc of urine

The Gilfillen and Gregg Antuitrin-S Skin Reaction Test: Two minims of antuitrin-S are injected intradermally The skin of the forearm is the location of choice. In a pregnant woman or in one who has aborted, but still retained some live decidual tissue, no reaction is noted. In a nonpregnant woman, or in one who has no retained decidual tissue, an erythematous area measuring from 7 to 35 mm. will appear around the site of injection within a few minutes

The Kantar, Bauer and Klawans Test: This test is based upon the observation that the female Japanese bitterling fish responds to an excess of estrogenic substance (female sex hormone) by elongation of the ovipositor from 2 mm. (normal size) to 15 to 25 mm within 36 to 72 hours.

A previously standardized fish is put into a two-quart bowl containing one quart of water. Four cc. of the urine to be tested is added, and the fish is observed at 24-hour intervals. A positive reaction is indicated by an elongation of the oviduct from its normal size of 2 mm. to 15 or 25 mm. After a positive reaction, the fish is put into a tank for recovery and left there for 2 or 3 weeks, the time required for regression to be completed.

Chemical Diagnosis of Pregnancy by Detection of Estrin in the Urine: This test, according to Schmulovitz and Wylie, consists in the extraction of the estrin (female sex hormone) from the urine with ether, and its detection by *coupling with diazotized paranitroaniline* to form a deep colored azo dye. The depth of color is then matched against a 33 per cent ferric chloride solution, the reading of which is recorded as the "ferric chloride number" (F.N.). A F.N. below 15 is considered negative, and above 25, positive.

Histidine Test for Pregnancy: This test is based on the exhibition of a positive histidine reaction with pregnant urine. Two reagents are used: (1) A bromine reagent consisting of 1 cc. of bromine, 100 cc of glacial acetic acid and 300 cc of distilled water, (2) an alkaline reagent, consisting of 10 Gm of ammonium carbonate dissolved in 90 cc of distilled water to which is added 200 cc. of ammonia; 2 to 5 cc of the bromine reagent is added to 5 cc of filtered urine, *then 3 cc of the alkaline reagent is added* and the mixture is thoroughly shaken and placed in a steaming bath for three minutes. The appearance of a mauve color, changing gradually to reddish purple, indicates a positive reaction. This test is not very reliable.

Tests for Viability of the Ovum: The viability of pregnancy may be determined by the hormone test when it is too early to determine it by other means.

In cases where any one of the accepted pregnancy tests was first positive and then became negative, there is an indication that the fetus is no longer viable.

When pregnancy tests were previously not made and pregnancy is suspected, the viability of the embryo or fetus may be determined, according to Spielman, Goldberger and Frank, by the female sex hormone blood determination. During pregnancy, the female sex hormone is found to be definitely increased. The finding of no increase of this hormone in the blood above the normal indicates that *pregnancy does not exist or that the* product of conception is dead.

Indication for Pregnancy Tests: Uterine pregnancy may, according to Goodale, be diagnosed by the Aschheim-Zondek test or the Friedman modification, one week after the first missed period. The Friedman method has given correct results in 98 per cent of the author's series of cases.

Diagnosis of ectopic pregnancy by this test is not quite satisfactory. It is positive in only about 50 per cent of the cases. When the test is positive in a case of supposed ectopic pregnancy, it is significant. When the test is negative, it does not rule out ectopic pregnancy.

The pregnancy test is markedly positive in cases of hydatidiform mole and chorionepithelioma. If the test remains *positive following surgery or radiation,* it indicates that there is a metastasis. If it becomes negative and remains negative, it indicates that the treatment has been successful and that there are no metastases. In the presence of hydatidiform mole and chorionepithelioma, blood

the filtrate is evaporated to dryness. The residue which remains is dissolved in water and when the solution is made slightly acid with H_2SO_4 and a drop of freshly prepared bromine solution is added, the iodine is oxidized to iodate. The addition of potassium iodide frees the iodine which is estimated by titration with 0.001 N sodium thiosulfate solution with starch serving as an indicator.

"The iodine content of the blood specimen secured five minutes after the injection of the Lugol's solution minus that of the preliminary control sample, is regarded as representing the maximum increment caused by the injected iodine and is consequently recorded as 100 per cent. With this value as a basis, the findings for the other samples are expressed accordingly. While the results so obtained represent the relative rather than the absolute iodine concentrations, they do provide an indication of the rate of disappearance from the circulating blood of the injected iodine in a specific time."

Watson found that in the normal, 9 to 23 per cent of the injected iodine remained in the blood stream six hours after its injection. In thyrotoxicosis and hyperthyroidism, all of the injected iodine was removed within six hours. In hypothyroidism the average quantity of iodine in the blood six hours after its injection was greater than normal.

Test for Pancreatitis and Hyperthyroidism

Loewi's Test: This depends upon an increase in the irritability of the sympathetic nervous system due to hyposecretion of insulin, and is performed as follows:

Two drops of 1:1000 adrenalin are instilled into the eye, and the pupil is examined 15 minutes later. Dilatation of the pupil is indicative of a lesion in the pancreas affecting the islands of Langerhans, *particularly if hyperthyroidism can* be excluded. If the pupil remains undilated at the end of 15 minutes, two more drops should be instilled and observation made 15 minutes later.

In hyperthyroidism, the administration of two to three drops in the eye causes prompt mydriasis which lasts from ten minutes to one hour or longer (SEE ALSO p. *1063*).

Test for Psittacosis

Complement Fixation Test: The patient's blood is used as the antigen (SEE: p. 1020).

Test for Rabies

The brain tissue of the rabid animal is examined for the *Negri bodies.* These *are round, oval or somewhat irregular* structures varying in size from 0.5 to 18µ (microns) and are usually found in the multipolar cells of Ammon's horn (hippocampus major). Their presence is positive proof that the animal had rabies.

Tests for Scarlet Fever

The Dick Test: This is utilized for determining the presence *of immunity.* The test consists of injecting intradermally 0.1 cc. of a culture of a specially prepared scarlet fever streptococcus solution. The reactions are observed at the end of 24 hours. An areola of from one to three centimeters in diameter is considered positive. A larger area which is markedly red and swollen indicates strong *susceptibility to scarlet fever.* A negative reaction indicates immunity.

Umber's Test: This is for the diagnosis of scarlet fever. Add two drops of a 30 per cent concentrated hydrochloric acid, 2 cm. of paradimethylamidobenzal-

dehyde dissolved in 70 cc. of water to a small quantity of urine. The appearance of a red color is said to be positive for scarlet fever.

The Schultz-Charlton Test: When scarlet fever antitoxin or convalescent serum is injected into the skin of a suspected scarlet fever patient, and blanching of the skin occurs at the site of injection, it indicates a positive reaction The injection of scarlet fever serum in the same patient's skin will not cause blanching.

Tests for Trichinosis

The Bachman Test: If the intradermal injection of a one per cent solution of powdered *trichina larvae* causes a well defined area of edema to develop within a week, the test is considered positive for trichinosis.

Muscle Biopsy: This may disclose the presence of the *Trichinella Spiralis.*

Tests for Tuberculosis

The Mantoux Intracutaneous Test (Mendel's Test): This consists of the intradermal injection of either 0 1 cc. of a 0.005 per cent or 1/200 mg. of a solution of old tuberculin or a 0 0002 mg of P.P.D (purified protein derivative) new tuberculin. 0 1 cc of a control solution consisting of 0 5 per cent phenol is injected a few inches above or below the test area A positive reaction consists of an area of swelling at the site of the tuberculin injection, 5 mm. or more in diameter, within 24 or 48 hours.

The Von Pirquet Test: The skin is slightly scarified over a small area; a small drop of old tuberculin is placed on and rubbed into this spot A control with glycerin sterile bouillon is made in a similar manner several inches distant from the test field. The excess of tuberculin is wiped off within five minutes. A positive reaction consists of the appearance, in 24 to 48 hours, of a red areola over the tuberculin treated area and none over the control.

When the reaction subsides, a brownish pigmented area may develop and last for several weeks.

Moro Test: This consists of rubbing into an area of the skin, about 1½ inches square, upon the anterior aspect of the chest or the inner side of the arm, about 0 5 Gm. of an ointment containing equal parts of tuberculin and sterile anhydrous lanolin. A positive reaction is indicated by the appearance of small papules over the treated areas in from 24 to 48 hours. The rash fades slowly.

Calmette's Eye Test: One or two drops of a 0.5 per cent purified old tuberculin solution is instilled into one eye. The development of conjunctivitis in the treated eye, in from 12 to 24 hours, constitutes a positive diagnosis. This test is now seldom used. In the presence of ocular disease, the Calmette test is dangerous.

The Patch Test: A small piece of linen impregnated with P.P.D. (purified protein derivative of tuberculin) is applied to the arm or forearm and permitted to remain *in situ* for 24 hours. On removal of the patch, the presence of an erythematous area denotes a positive reaction.

Hypodermic Injection Test (The Tuberculin Test): This is probably among the earliest tests performed for the diagnosis of tuberculosis and is at present displaced by the Mantoux, Von Pirquet and Patch tests. This test consists of the hypodermic injections of 0.01, 0.1, 1, 2, 5 and 10 mg. of old tuberculin successively three or four days apart,

the filtrate is evaporated to dryness. The residue which remains is dissolved in water and when the solution is made slightly acid with H_2SO_4 and a drop of freshly prepared bromine solution is added, the iodine is oxidized to iodate. The addition of potassium iodide frees the iodine which is estimated by titration with 0.001 *N* sodium thiosulfate solution with starch serving as an indicator.

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Umber's Test: This is for the diagnosis of scarlet fever. Add two drops of a 30 per cent concentrated hydrochloric acid, 2 cm. of paradimethylamidobenzal-

ing drop method after a 2-hour incubation. Under the oil immersion lens, the positive slides will show clumped, motionless masses of bacilli.

The agglutination tests are employed for the detection of typhoid fever, paratyphoid fever, tularemia, undulant fever, etc. In these cases the known bacteria are brought in contact with a suspected or unknown serum. The agglutination or clumping of the bacteria by the serum in high dilutions identifies the disease

Test for Pancreatic Disease (Other Than for Diabetes)

Serum Amylase: The normal values of serum amylase are between 70 and 200 units. In acute inflammation or obstruction of the pancreas the amylase values may reach 3000 units. An increase in the serum amylase is occasionally found also in those suffering from affections of the gastrointestinal tract adjacent to the pancreas, *i e.*, cholecystitis, peptic ulcer, gastritis and some liver affections. Moderately increased amylase values are at times also found in mumps, typhoid fever and other infections.

Test (Somogyi's Method): To 1 cc. of blood serum or plasma is added a mixture of 5 cc. of 1.5 per cent cornstarch solution and 2 cc of 1 per cent sodium chloride solution and this is incubated for 30 minutes at 104° F. (40° C.). Then to this are added 1 cc. of 5 per cent solution of copper sulfate and 1 cc. of 7 per cent solution of sodium tongstate. This mixture after shaking is filtered and is analyzed for sugar. Correction is made for the presence of glucose in the serum and substrate. The result is expressed in milligrams of glucose liberated per 100 cc. of serum. Two hundred units of amylase is represented by 200 mg. of glucose liberated per 100 cc. of serum.

Urine Amylase: The normal values of urine amylase is 3 to 32 units. In pancreatic disease the urine may contain 200 or more units. This test depends upon *the quantity of urine capable of neutralizing* 5 cc. of a 1 per cent starch solution.

Serum Lipase: Normally the blood serum contains very little, if any, lipase. In *pancreatic disease* the lipase values may be as high as 10 units or more per cc. of serum. The technic of this test is involved and requires considerable technical skill and laboratory facilities. An increase in the serum lipase is at times also found in liver disease and carcinoma of the ampulla of Vater.

Tests for Kala Azar

A positive diagnosis of kala azar by laboratory methods can only be made when the Leishmania donovani are found in blood smears or in smears of material obtained by puncture of the liver, spleen, sternum or infected glands A measure of corroboration in the diagnosis of kala azar and schistosomiasis in the presence of suggestive clinical signs may be had by one of the following three simple tests

I **The Water Test:** To 0 6 cc. of freshly distilled water in a small test tube add 0 02 cc of freshly drawn blood and shake gently. Allow this mixture to stand for five minutes. If it becomes cloudy, or if at the end of 15 minutes there occurs a definite sediment the test is considered positive.

II **The Formalin Test:** To 1 cc. of clear serum add one drop of 30 per cent formalin solution and shake until well mixed. Allow this to stand for 15 minutes. The test is considered positive when the mixture solidifies to the consistency of the white of a hard-boiled egg This reaction is usually seen in old cases of kala azar.

after the patient's temperature has been determined. A rise of 1° F. within 8 to 12 hours after an injection constitutes a positive reaction. If the temperature rise is noted after any one of these injections, further injections are not necessary. If no rise in temperature occurs after the largest dose, the test is considered negative.

Tests for Cerebrospinal Tuberculosis

The Levinson Test: This is based upon the finding that the ratio between the alkaloidal precipitate formed by sulphosalicylic acid and the metallic precipitate formed by mercuric chloride is altered. A positive reaction is indicated when the mercuric chloride precipitate is three times as great as that formed with sulphosalicylic acid. In the normal, the mercuric chloride precipitate forms slowly and is feathery, while the sulphosalicylic acid precipitate starts forming rapidly and is heavy and compact.

Tryptophan Test (Lichtenberg): When the cerebrospinal fluid in a test tube is slowly brought in contact with the reagent and a violet ring is formed at the junction, the reaction is considered positive. The reagent in this test consists of concentrated hydrochloric acid (15 to 18 cc), two or three drops of a two per cent formaldehyde solution, and 1 to 2 cc. of 0 06 per cent sodium nitrite solution. In the absence of tuberculous meningitis, there is either no ring at the point of contact, or a brown ring is formed.

Tests for Undulant Fever

This gives a positive agglutination reaction in high dilutions. (See: Agglutinations Tests, p. 1019 and next column.)

Burnet Intradermal Test: A small quantity of a filtrate of a 20-day bouillon culture of micrococcus melitensis is injected intradermally If *positive, there* will appear within six hours after the injection, an area of redness and swelling at the point of inoculation, and at times also a rise in temperature and headache

Agglutination Tests

Agglutination tests may be performed by two methods, the macroscopic and the microscopic.

The Macroscopic Method: In this method the blood serum is placed in each of seven test tubes; the first tube is undiluted and each of the following tubes is progressively diluted so that they contain 1 : 10, 1 : 20, 1 . 40, 1 : 80, 1 : 160, and 1 · 320. To each tube is now added 0 5 cc. of the suspension of killed bacteria for which the test is performed. This doubles the dilution of the serum in each of the tubes, each having the following dilutions, 1 : 20, 1 . 40, 1 · 80, 1 : 160, 1 . 320, and 1 : 640 These tubes are thoroughly shaken and then placed in an incubator for 8 to 12 hours Positive reactions consist of the formation of a sediment made up of agglutinated bacteria at the bottom of the tube; the rest *of the tube contents remain clear.* The tubes in which the agglutinations occur indicate the degree of concentration. Thus, concentrations may be positive in 1 : 40, 1 : 60, 1 : 320, etc., the higher the concentration, the more positive is the reaction.

The Microscopic Method: A series of dilutions of the serum is arranged as in the macroscopic test. A droplet of each diluted serum is placed upon a slide and to each droplet of diluted serum is added a loopful of a 24-hour-old bouillon culture of *the organisms to be tested.* Each is examined according to the hang-

SECTION 16

Parasitology

III. The Antimony Test: In positive cases a heavy precipitate is formed when two drops of the patient's serum is added to 1 cc. of 0.5 per cent solution of urea stibamine or other pentavalent antimony compound.

These tests may also be positive in bacterial endocarditis or in other conditions associated with a marked increase in serum globulin.

The Congo Red Test for Amyloidosis

This test is based on the affinity of congo red for amyloid.

Test: 0.25 cc. of 1.5 per cent aqueous solution of congo red per Kg. of body weight is injected intravenously. The maximum amount is not to exceed 18 cc. About 10 cc. of blood is withdrawn (from one of the veins not previously used) after four minutes and after one hour. These specimens are centrifuged and the separated plasmas are compared with each other in a colorimeter. The four-minute specimen serves as a standard and is considered as containing 100 per cent of the dye.

The one-hour specimen is the indicator as to the amount of dye absorbed by the tissues and therefore cleared from the blood. Normally the rate of absorption from the blood is slow and the one-hour specimen may have cleared only from 10 to 30 per cent of the dye. In amyloidosis the blood is cleared rapidly so that the one-hour specimen may contain no dye or only a small amount A clearance of over 60 per cent is suspicious of amyloidosis

The Heterophile Antibody Test

This test depends upon the agglutinins and hemolysins in the blood having an affinity for other antigens or antibodies besides those for which they are specific.

Paul and Brunnel in 1932 reported that about 90 per cent of patients suffering from infectious lymphocytosis (glandular fever, infectious mononucleosis) possess, in their blood serum, heterophile antibodies in the form of agglutinins for sheep red corpuscles, in a titer of 1 to 32 or higher.

Normal persons may show a positive seroreaction in a titer of 1 to 8, and individuals to whom horse serum was administered may show a positive reaction in dilutions of 1 to 64 or higher

In infectious mononucleosis, during the first week or 10 days, agglutination reactions may be present in low titer; after the second week the titer may be 1 to 256 or higher, usually remaining high up to the fifth week, when it falls off rapidly In a small number of cases *the heterophile antibody test is negative* This is more likely to be found among *very young children*

A temporary positive Wassermann reaction may be elicited in a small percentage of cases during the height of the disease; that is, during the period in which the agglutinins are present in high titer.

The technic of the heterophile antibody test is that of the agglutination test (SEE: p. 1062), except that 0.5 cc. of a 2 per cent suspension of washed packed sheeps' corpuscles is used *instead of* 0 5 cc. of a suspension of killed specific *bacteria*

To make the heterophile antibody test more specific for glandular fever (infectious mononucleosis) and exclude nor [illegible] e
to [illegible] y
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ferential absorption *tests" with guinea* pig kidney and ox cells.

CHAPTER XXXVIII

Parasites and Parasitic Infections

While a fairly large number of parasites causing specific diseases are found in the blood and in other tissues of man, the greatest majority of parasites have their habitat within the gastrointestinal canal and may cause local or systemic diseases.

Animal parasites affecting man are classified according to their structures into three divisions. Some of these classes are further subdivided into behavior and structural groups, each of which is responsible for a specific type of disease. *The three main divisions are Spirochetes,* Protozoa, and Metazoa. The last group includes Trematodes or Flukes, Cestodes or tapeworms and flatworms, Nematodes or roundworms, Insects and other Arthropods.

Spirochetes (Spirochaetales)

The spirochetes really belong to the order of Schizomycetes, an intermediate between bacteria and protozoa. They infest the solid tissues, blood, spinal fluid and occasionally the urine The subgroups of this division are: (*a*) The *Treponema pallidum,* causing syphilis (SEE: p. 56); (*b*) the *Treponema petenue,* causing yaws (SEE: pp 56 and 143); (*c*) the *Spirillum minus,* causing ratbite fever (SEE. p 56); (*d*) the *Spirochete borelia,* causing relapsing and tick fevers (SEE· p. 56), and (*e*) the *Leptospira icterohaemmorhagiae,* causing Weil's disease (SEE: p. 56).

The Protozoa

The protozoa belong to the lowest animal kingdom and are unicellular organisms. They are subdivided into four groups.

(*a*) **The Sarcodinia Possessing Pseudopodia:** To this group belongs the Endamoeba histolytica (Entamoeba histolitica) which causes ambiasis or amebic dysentery (SEE· p. 57), and the nonpathogenic group of amebae, *i. e.,* ameba coli, endolimax nana, iodamoeba butschlii, and dientamoeba fragilia. The habitat of the ameba group is the colon They enter the body with infected food or drink containing the organisms or *their cysts.*

(*b*) **The Sporozoa:** To this group belong the four species of plasmodia responsible for malaria (SEE· p. 57 and 1089). These are the *plasmodium vivax,* causing the benign tertian type of malaria; the *plasmodium malariae,* causing the two quartan types; the *plasmodium falciparum,* causing the estivo-autumnal, tropical quotidian, malignant tertian and subtertian types, and the *plasmodium ovale,* which resembles the vivax species The plasmodia are transmitted to man by an Anopheline mosquito (SEE: p 1087) and may also be transmitted by injecting blood from a malarial patient into the circulation of a normal individual.

Others of the sporozoa group are the *coccidia* and the *sarcosporidia* which are prevalent in herbivorous animals The *sarcocystis lindermanni* causes *sarcosporidiosis* in man. These organisms are found in the striated muscle fibers of the tongue, larynx and myocardium. Among the sporozoa group may also be mentioned several species of *toxoplasma*

developing irregular intermittent fever; the periods of remission are variable. There are headache and progressive weakness. The skin shows a patchy erythema, localized edema and hyperparethesia The lymph glands enlarge and are tender; the spleen and liver gradually enlarge and anemia develops The second or cerebral stage may develop within several months *or a year or more* The outstanding symptoms are increased weakness, mental dullness, and disinclination for exertion The face is puffy and carries a vacant expression The gait is slow and shuffling There develop tremors, headaches, and somnolence from which the patient may be aroused with difficulty *Paralysis of the lips, nuchal rigidity and maniacal* symptoms are terminal manifestations The blood may contain the organism but in small numbers; animal inoculation may aid in the diagnosis

Chagas' Disease (*South American Trypanosomiasis*): This is a form of sleeping sickness found chiefly among infants and young children in South America It is caused by the *Trypanosoma cruzi* which is transmitted by a reduviid bug of the genus *Triatoma*

The clinical manifestations are divided into two stages, acute and chronic During the acute stage, the organisms are found in the blood The symptoms are fever, myxedematous swellings, listlessness alternating with irritability, enlargement of the lymph glands, spleen, liver, and thyroid The thyroid gland becomes especially large and hard During the chronic stage, the organisms are found in the tissues, the symptoms are severe and depend upon the structures involved These may be cerebral, cardiac, adrenal (Addisonian) manifestations, etc The thyroid gland is large and stony hard, causing various degrees of myxedema.

Kala Azar (Visceral Leishmaniasis, Dumdum Fever, Black Fever): This is an infectious disease running a protracted course; it is characterized by huge enlargement of the spleen, moderately enlarged liver, irregular fever, and anemia with leukopenia. The disease is prevalent in Eastern India, Northern China, and is also met with in the Sudan, West Africa, Iraq, the countries bordering the Mediterranean and in South America

Kala Azar is caused by the protozoon Leishmania donovani which may be carried by a bedbug (*cimax hemipterus rotondatus*) and possibly also by a species of sand fly (*phlebotomus argentipes* or other species)

The *Leishmania donovani*, on entering *the body*, are taken up by the cells of the reticuloendothelial system where they develop, causing the cells to burst and to discharge the parasites into the blood stream. The entire reticuloendothelial system proliferates and infected plasma cells are found in the spleen, bone marrow, liver (Kupffer cells) and throughout the body where reticuloendothelial tissue is normally found. While the parasites are most numerous in the reticuloendothelial system many are also found in various other organs

Symptoms: The onset is insidious with fever which may be continuous or remittent, and it may have a double or triple rise in 24 hours Chills may accompany each rise of temperature The splenic enlargement becomes palpable after the first month; by the end of the sixth, the spleen is huge The liver also becomes enlarged There are progressive weakness, emaciation and anemia. The leukocytes may fall from 4000 to 1000 per cmm The blood platelets are low,

which cause the rare disease of childhood, "Toxoplasmosis" The organisms are usually found in the brain, spinal cord, choroids, heart and in the skeletal muscles, thus causing toxoplasmic encephalitis and systemic infection

Toxoplasmic Encephalitis: Sabin[1] reported two cases of toxoplasmic encephalitis in children One, a boy, age 6 years, died within one month after onset. The outstanding symptoms were headache, convulsions and vomiting The temperature ranged between 99 6° and 101° F. during the first 20 days, and subsequently rose to a higher level and reached 108.4° F. just before death

The other case was a boy, age 8 years, who developed atypical encephalitis and recovered in nine days Both cases had toxoplasma in the spinal fluid and toxoplasma were isolated from guinea pigs inoculated with the patients' spinal fluid

Systemic Toxoplasmic Infection in Adults: Pinkerton and Henderson[2] reported two such fatal cases. In each case there was a history of the patient having picked some ticks from off his body The clinical manifestations were fever, adenopathy, a maculopapular eruption involving the entire body but sparing the palms of the hands, soles of the feet and scalp Both cases showed signs of lung involvement and general toxemia. The toxoplasma were recovered from the lungs and were isolated from guinea pigs injected with the patient's blood.

(c) The Parasitic Infusoria Group (*ciliated protozoa*): To this group belongs *Balantidium coli*, which cause Balantidiasis. The organisms are found in the colon of man and may cause chronic diarrhea with more or less blood in the stool. The parasites are prevalent in the intestines of the pig and wild rat The *Balantidium minutum* and the *Nyctotherus faba* are infusoria which rarely invade the human intestinal mucosa

(d) The Mastigophera or Flagellates: This group includes *trypanosoma gambiense* and *trypanosoma rhodesiense*, which cause trypanosomiasis, or sleeping sickness; the *trypanosomiasis cruzi*, which cause Chagas' disease; the *Leishmania donovani*, which cause Kala Azar; the *Leishmania tropica* which cause Cutaneous Leishmaniasis and Mucocutaneous Leishmaniasis (American Leishmaniasis). These invade the blood stream, the glands and other structures of the body. In addition there is a group of flagellates that invades the intestines and may cause diarrhea or other minor symptoms

Trypanosomiasis (Sleeping Sickness): There are two types of sleeping sickness found in Africa. *The mild type*, found in the Belgian Congo, Uganda and Tanganyika Territory, is an infection by the *trypanosoma gambiense* carried by two species of Tsetse fly, *Glossina palpalis* and *Glossina tachinoides*. The disease runs a relatively mild course, exhibiting a moderate intermittent fever, some erythematous skin areas, palpable lymph glands, localized edema, moderately enlarged spleen, and drowsiness.

The severe type is found chiefly in Nyasaland and Rhodesia, and is caused by *Trypanosoma Rhodesiense*, which is transmitted by the bite of the Tsetse flies, *Glossina Morsitans* and *Glossina Swynnertoni*. This type runs a shorter but severer course. The clinical manifestations may be divided into two stages The first stage is marked by a slowly

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The subvarieties of trichomonae are *Trichomonas fecalis, Pentatrichomonas ardin delteili* and *Chilomastix mesnili* (Macrostoma mesnili, chilomastix devainei, tetramitus mesnili) Their natural habitat is in the colon and they may be recovered from the stool. Other and less commonly found flagellates in the intestines are: The *Embadomonas intestinalis* (*Washkia intestinalis*), a very small flagellate; the *Enteromonas hominis; Fonseca,* and *Craigia hominis.* Intestinal flagellates may cause diarrhea, cramps, digestive disorders, occasionally anemia, and often their presence is unsuspected.

The Metazoa

Trematodes or Flukes: The flukes occurring in man are small nonsegmented flat, usually tongue or leaf-shaped, organisms well supplied with suckers. Most of the flukes are hermaphroditic, a few of the species, those infecting the blood, are unisexual Flukes may be classified according to their habitat in man as those infesting the intestines, those infesting the liver, those infesting the lungs, and those found in the circulating blood.

(*a*) ***Intestinal Distomiasis:*** The *Fasciolopsis buski,* commonest of the flukes, resides in the small intestine, and occasionally in the stomach of both man and pig. The life cycle of this as of other flukes, according to Barlow and to Nakagawa, is as follows· The eggs discharged with the feces in water are hatched as miracidiae in three or more weeks. They then penetrate various species of snails and produce generations of rediae; these develop into cercariae and as such leave the snail and become encysted on aquatic plants When these plants are eaten raw, the encysted cercariae find their way into the small intestine where they mature into adult flukes.

Symptoms Intestinal distomiasis is divisible into three stages:

(1) The period of latency in which there are no characteristic symptoms except perhaps some unaccountable weakness.

(2) The period of diarrhea in which there is abdominal pain, more or less diarrhea and a peculiar transparency and puffiness of the skin due to subcutaneous edema.

(3) The period of edema in which there develop ascites and edema of the genitalia, and of the lower extremities. This later spreads to the face and lungs Cardiac insufficiency becomes marked. The skin is dry, harsh and icteroid, and the tongue is dry. The temperature is usually subnormal. The disease is widespread in southern and western Asia, and the nearby Pacific Islands Other of the intestinal flukes smaller than the *Fasciolopsis buski,* which cause enteritis and other manifestations of intestinal distomiasis, are indigenous to Africa, Asia, and to some of the Pacific Islands These are the *Watsonius watsoni* of Northern Nigeria, the *Heterophyes heterophyes* of Egypt, the *Gastrodiscoides hominis* of India and Assam, the *Heterophyes nocens* of Japan; the *Metagonimus Yokagawai* of Formosa, Japan and China, the *Echinostoma ilocanum* of the Philippines, and the *E Malayanum* of the Malay States.

(*b*) ***Hepatic Distomiasis:*** Liver flukes usually invade the bile ducts and may also travel to the pancreatic ducts. The commonest of this group is the *Clonorchis sinensis* This parasite is prevalent in the Eastern Asiatic countries and affects man and fish-eating animals. Massive infection with this

and bleeding and coagulation time are prolonged. The serum *globulin is increased* and the albumin is decreased. The *abdominal veins enlarge* and there may be edema of the legs. Blood *culture and spleen and liver puncture will reveal the flagellated* protozoon (For presumptive tests see p. 1063.)

Cutaneous Leishmaniasis (Oriental Sore, Aleppo Boil, Delhi Boil): This disease is found in India, Persia, Palestine, Africa, in many of the Mediterranean countries, and in South America. The lesion appears on any exposed part of the body, usually upon the face or on the upper extremities, as a *small red* papule which gradually enlarges. The center softens and breaks down, *forming* a round soft-edged ulcer. This may heal slowly, in from three months *to a year*, leaving a slightly depressed scar. One attack confers *immunity. The protozoal* parasite, *Leishmania tropica*, may be recovered from the *fluid obtained by puncturing* the lesion. The insect vector is a phlebotomus sand fly.

American Leishmaniasis (Mucocutaneous, Nasopharyngeal or Brazilian Leishmaniasis, or Espundia or Forest yaws): This type usually affects the mucous membranes of the nose and throat, though the lesion may appear on any exposed part of the body. When the lesion invades the mucous surfaces it produces a fungating ulcer which in*filtrates the deeper structures, causing* destruction of the nose, part of the face *and occasionally the larynx and pharynx.* When the lesion occurs upon the skin, *it resembles "Oriental Sore."*

(*b*) **Flagellate Diarrhea** (Giardia *Lamblia, Giardia Intestinalis*, Lamblia Intestinalis): Flagellates invade the du*odenum and jejunum.* The organisms and their cysts may be recovered from *the stool. Trichomonas hominis* (Trichomonas intestinalis, Trichomonas confusa) are usually found in the colon

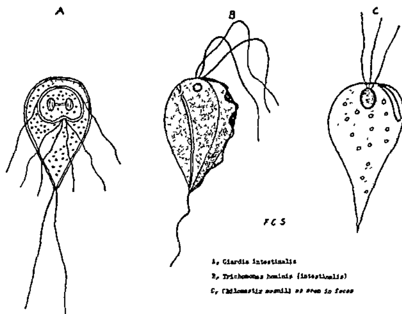


Fig. 1.

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trematode will cause jaundice, anemia, ascites, edema, cachexia, bloody diarrhea and epistaxis. Other flukes, commoner in animals than in man, are the *Fasciola hepatica,* found in sheep; *Dicrocoelium lanceatum* found in herbivorous and omnivorous animals; *opisthorchis felineus* found in the gallbladder and bile ducts of cats, dogs, pigs, foxes and at times, in man, and *opisthorchis caninus* found in wild dogs.

Life Cycle of the Liver Flukes The eggs are taken up by a snail (the *parafossarulus striatulus,* or the *Bithynia fuchsiana*), which is the intermediate host. After hatching, the cercariae escape from the snail and enter the bodies of certain fresh-water fish and become encysted beneath the scales or in the deeper tissues where they may survive for many years. The adult worm develops in man, dog, cat or other animal which has eaten the infected fish

(*c*) ***Pulmonary Distomiasis:*** The best known of the group of flukes causing pulmonary distomiasis are the *Paragonimi westermanu* They are found chiefly in Eastern Asia and the Pacific islands. The worms are for the most part encysted and are lodged not only in the lungs but occasionally also in the intestines, pancreas, liver, spleen, bladder, epididymis, prostate and choroid plexes of the brain (Tyzzer and Smillie). Fever, cough, and bloody expectoration are common symptoms when the lungs are invaded Involvement of other organs will cause systemic and local reaction referable to those organs.

Life Cycle of the Lung Flukes· The eggs develop in water as ciliated embryos. As such they are taken up by a certain species of snail which acts as the first intermediate host, The second intermediate hosts are many species of fresh-water crabs and crawfish which, when eaten by man or animal, transmit the embryos to them On entering the stomach of their final host the embryos are liberated from their cysts, penetrate the intestinal mucosa and work their way through the peritoneal cavity, diaphragm, pleurae and lodge chiefly in the lungs and occasionally in other organs as adult worms. The adult worms become encysted and lay their eggs which are discharged with the feces or sputum and which may also be recovered by aspiration

(*d*) ***Hemic Distomiasis, Schistosomiasis, Bilharziasis:*** The three species of blood flukes responsible for this condition are: The *Schistosoma hematobium;* the *Schistosoma Mansoni,* and the *Schistosoma Japonicum* These worms, unlike the other flukes, are of separate sexes

The *Schistosoma hematobium* invade the portal system, the mesenteric vein, the hemorrhoidal veins and plexuses but lodge chiefly in the veins of the urinary bladder and the bladder wall Hematuria, renal calculi, ureteral obstruction and infection are among the common symptoms There are also eosinophilia and occasionally dysentery with tenesmus This infection is prevalent in North Africa and the Near East

The *Schistosoma mansoni* infection occurs in Africa, the West Indies and certain parts of South America This worm invades chiefly the mesenteric veins and causes chronic dysentery, colic and emaciation.

The *Schistosoma Japonicum,* causing Katayama disease, invades chiefly the walls of the intestines, and less frequently the liver, spleen, lungs and brain, causing severe diarrhea, dysentery, painful

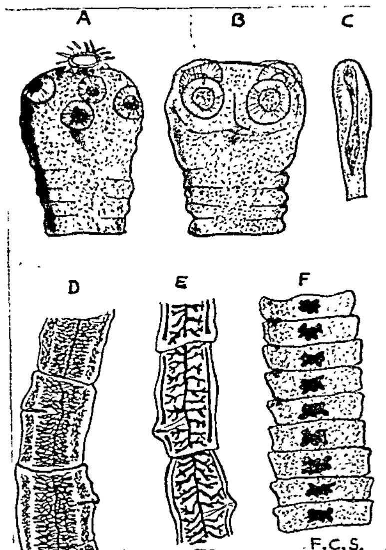


Fig 2—*A*, Head of Taenia solium, *B*, head of Taenia saginata, *C*, head of Diphyllobothrium latum, *D*, segments of Taenia saginata, *E*, segments of Taenia solium, *F*, segments of Diphyllobothrium latum.

enlargement of the liver and spleen, dropsy, and anemia and cerebral symptoms.

The Life Cycle of the Blood Fluke The eggs are hatched in water and are taken up by certain snails The cercariae that develop in the snails escape in the water as free-swimming larvae and may enter the bodies of man or animal by either the alimentary canal or the skin Within the body of their final host they develop into adult worms

The Cestodes (Tenia or Tapeworms): Tapeworms also infect men through an intermediate host and they occur in two forms. One form resides in the small intestines of man, in the adult state causing Intestinal Teniasis,

the parasites having entered the intestines as embryo with the flesh of a specific host. The other form is found in the muscles or other tissues of man in a developmental stage causing somatic teniasis.

Intestinal Teniasis: The adult tapeworms residing in the human intestines are:

The Diphyllobothrium Latum (Dibothriocephalus Latus, Fish Tapeworm, or Broad Tapeworm) · This is the longest and broadest of the tapeworms. It may attain from 3 to 13 meters (10 to 40 feet) or more in length and from ⅓ to ½ of an inch in breadth at its broadest end. It gradually tapers down towards its long thin neck, reaching its narrowest part at the almond-shaped head. The segments are broad and short; each segment contains a centrally situated tortuous ovarian rosette where is also found its sexual orifice. The worm is hermaphroditic The *diphyllobothrium latum* is commonly found in the Baltic Sea region, in Japan, in Turkestan, Poland, Switzerland, Rumania and less frequently in the United States and Canada. Before reaching the adult stage in man it passes through two intermediate hosts When immature eggs from human stool enter a fresh water stream they undergo some development and when ingested by a cyclops or other crustacean, further develop into procercoid larvae. These larvae, when swallowed by the pike or other fresh water fish, develop into the plerocercoid stage and invade their tissues. When the uncooked or insufficiently cooked flesh of the infested fish is eaten by man or by certain animals the larvae finally develop into adult worms and inhabit the intestines of their hosts. Infection with this tapeworm may cause no symptoms; occasionally, however, it may cause a severe type of hyperchromic macrocytic anemia resembling primary pernicious anemia. Segments of various lengths and ovae may be found in the stool

Tenia Saginata (Beef Tapeworm): This worm does not attain the length or breadth of the fish tapeworm; it may measure from 2 to 10 meters (6 to 30 feet) in length and about ¼ inch in breadth; the segments are longer and thinner than those of the fish tapeworm. The genital pores alternate and are not centrally placed The head possesses four suckers but no hooklets. This tapeworm may be found in human intestines wherever beef is eaten The *cisticercus bovis* (encysted larva) is found in muscles of infected cattle, particularly in the pteryoid muscles. When raw or rare infected beef is eaten by man the larva on reaching the human intestines develops into an adult worm. Individual segments or proglottids of the worm are frequently found in the stool or may lodge in the rectum, these often exhibit a crawling motion, thus resembling individual worms The symptoms produced by this worm are vague; there may be some abdominal pain, indigestion, excessive appetite or anorexia, and vomiting. In most instances the presence of the parasite is first manifested when found in the stool

Tenia Solium (Pork Tapeworm): This worm is smaller than the other two preceding types; it may measure from 2 to 3 meters (6 to 10 feet) in length The head is globular and possesses four suckers, a restellum and a double row of hooks. The proglottids are bisexual The adult worm resides in the intestines of man. The larvae (*cysticercus cellulosae*) are found in the striated muscles of the pig, wild boar, brown bear, stag.

dog, cat, monkey, and, at times, also in man (SEE: p. 1077). This tapeworm is transmitted to man in two ways. The common mode of infection in which the adult worm eventually lodges in the intestines is acquired by eating insufficiently cooked "measly pork," or pork sausage made of pork infected with cysticerci. Pickling and smoking do not kill the cysticerci. The other and less frequent source of human infection where cysticerci lodge in the tissues and remain there as embryos (Somatic Teniasis) is caused by autoinfection This may be brought about in two ways · (1) By the regurgitation of segments of the adult worm from the intestine into the stomach during vomiting, and (2) by the transmission of oncospheres through food which came in contact with hands or clothing contaminated with infected human feces For the worm to reach its adult stage in man, the embryo must undergo further development in the hog or other intermediate host Infection with this tapeworm is found most frequently where uncooked pork products are consumed and where sanitary regulations are lax

Hymenolepis Nana (Dwarf Tapeworm) · This tapeworm is common in Southern United States, in Sicily, Italy and other parts of Southern Europe and in India It measures from 2 5 to 4 cm (1 to 1½ inches) in length It inhabits the small intestines of man and, according to Grassi, does not require an intermediate host for its development The eggs hatch out in the intestines and there develop into embryos The embryos penetrate the mucosa of the intestines and further develop into cercocysts, as such they attach themselves to the villi of the intestines where they develop into mature worms. The symptoms produced by infestation with this worm are similar to those produced by other tapeworms, *i. e.*, vague digestive disturbance, irritability, weakness, etc. Examination of the stool may identify the worm, its eggs or its cercocysts. A similar tapeworm, *Hymenolepsis fraterna,* is found in rats. Larval forms of this worm may also develop in insects that ingest the eggs.

Somatic Teniasis: The tapeworms residing in their developmental stage in *the tissues of man are:*

Diphyllobothrium Mansoni (Dog or Cat Tapeworm): In its plerocercoid stage it is known as *Sparganum mansoni* Its life cycle is similar to that of the diphyllobothrium latum except that it does not occur in man in its adult form Its life cycle is as follows: The adult worm is found in the intestine of dogs or cats; the eggs in the feces of an infected animal are ingested by small crustaceans or by cyclops leuckarti, the first intermediate hosts in whom they develop into procercoid larvae. This host may in turn be swallowed by the second intermediate host which may be man or other mammal, bird, snake, or frog.

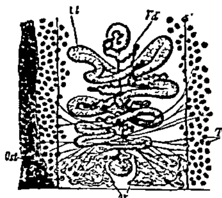


Fig 3—Dorsal or male aspect of a proglottid of Diphyllobothrium latum. *T*, Testes *Vd*, Vas deferens.

In the second host the larvae are liberated, penetrate the stomach and find their way under the peritoneum and thence migrate to the somatic muscles, the pleurae, the eyes, the genital tract and under the skin When infected tissue is ingested by dogs or cats, adult worms develop in their intestines. The local application of an infected frog to a superficial ulcer has, according to Joyeus and Houdemer, caused this infection to appear in man. The symptoms are pain, local swelling and edema.

Sparganum Proliferum: This is prevalent in Japan. The cerci cause superficial nodules and may affect various tissues Elephantiasis may result from their invasion of the lymph channels.

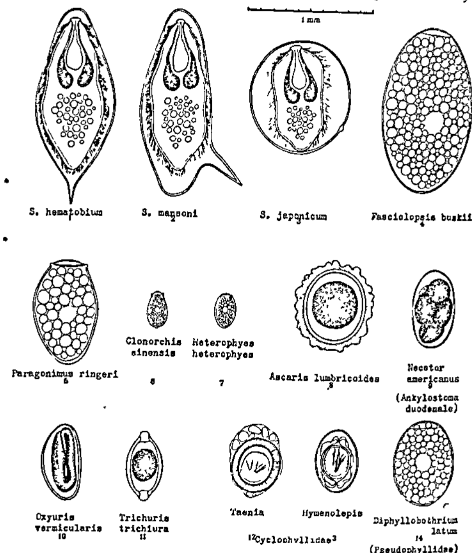


Fig. 4—Ova of the commoner human parasites.

Cysticercus Cellulosae: The larval stage of tenia solium (pork tapeworm), as previously mentioned, may invade various tissues of man who may or may not be infected by the adult parasite.

The symptoms produced by this worm are more severe when the larvae invade the tissues forming cysticerci cellulosae than when the adult worm resides in the intestines. The *cysticerci cellulosae* may occupy any organ or tissue of the body These cysts have been found in the brain, the eye, the heart, the lungs, the liver, the abdomen, the striated muscles, and in the subcutaneous tissue. They may occur in large numbers The clinical manifestations depend upon the site of the invasion Irregular fever, muscle pain, headache, anemia, and transient eosinophilia are general findings When the brain is involved there may be local or general convulsions and other signs suggesting encephalopathy Involvement of the subcutaneous tissue is characterized by the formation of palpable cysts varying in size from a pea to that of a hazel nut. These may be found all over the body, but chiefly in the upper half Massive infection, particularly in vital organs, may cause death.

Tenia Multiceps (Coenurus Cerebralis) · The cysts of this canine tapeworm usually affect the brain of goats and sheep They may also invade the brain of man, causing aphasia and epilepsy.

Echinococcus Granulosus (Dog Tapeworm): *In their larval or cystic stage these cause Echinococcus or Hydatid cysts* The adult tapeworm measures 2 5 to 6 mm in length. It inhabits the intestines of dogs, jackals and wolves. The intermediate hosts are sheep, cattle and pigs The larvae are transmitted to man by the drinking of water or by the eating of raw vegetables contaminated with infected canine feces In man, the embryo penetrates the intestinal mucosa, invades the blood stream and may lodge in the liver, lungs, brain, kidneys, bones and muscles, causing Echinococcus or Hydatid disease.

Echinococcus or ***Hydatid Disease*** is characterized by the formation of cysts which are often large and contain many brood capsules and scolices. The liver is the organ most frequently affected. *Occasionally an echinococcus cyst* may undergo secondary infection and suppurate. The disease may be acquired during childhood and may remain symptomless for many years.

Diagnosis· Since echinococcus disease is characterized by the formation of large cysts, the clinical findings of a large liver containing a cystic mass or evidence of cyst in the lungs, bone or brain, accompanied by weakness, and other signs of chronic ailment in one who had been in close contact with dogs, particularly in rural districts, should arouse suspicion of this infection. A positive diagnosis may be made by obtaining a positive complement-fixation test and precipitin reaction, and a positive skin test made with the fluid obtained from hydatid cysts of cattle. The blood smear will reveal marked eosinophilia.

Nematodes (Roundworms): Roundworms are cylindrical-shaped worms varying in length, thickness and habitat.

Ascaris Lumbricoides (Intestinal Roundworm): This, the commonest of all worms affecting man, resembles the common earthworm These worms infest the small intestines, especially of children; occasionally they may migrate to various places, *e. g.*, into the stomach and be vomited up- or downwards and pass through the rectum and, rarely, they may enter the gallbladder and bile ducts, causing biliary obstruction. They have been known to enter the larynx, lungs, nose and Eustachian canal. These

worms are pinkish or reddish yellow in color, measuring from 15 to 40 cm. in length and about 0.5 cm in thickness. The female is larger than the male. The roundworm is indigenous to all countries, but is more prevalent in warmer climates and in rural districts.

Symptoms: Their presence may not be suspected until found in the stool. At times they may cause cramps, nervousness, irritability, and, when the larvae or the adult worms invade unusual sites, they may cause local manifestations. The ova passed in human feces develop in night soil The fertilized eggs, when swallowed with contaminated water or food, develop into larvae, penetrate the bowel wall and migrate with the circulation into the liver, lungs, etc ; they pass up the trachea to the larynx, and are swallowed into the stomach and finally reënter the intestines where they develop into adult worms.

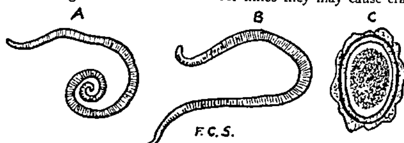


Fig. 5—Ascaris lumbricoides *A*, Male, *B*, female, *C*, egg.

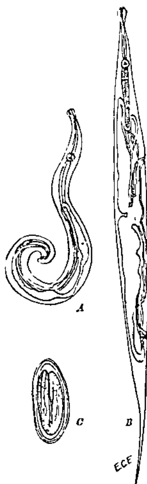
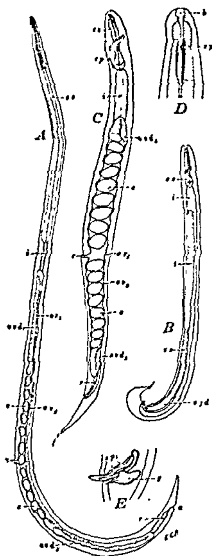


Fig. 6—Enterobius vermicularis. *A*, Adult male, ×16; *B*, adult female, ×16; *C*, egg from gravid female, with fully developed larva, ×280 (Faust's "Human Helminthology," Lea and Febiger).

A variety of this worm in both the embryonic and adult stages is found in the domestic pig.

Oxyuris Vermicularis (*Enterobius Vermicularis, Ascaris Vermicularis*) (Seat-, Pin-, Thread-, or Manworm): These worms inhabit the lower colon and especially the rectum, and are found most frequently among children in whom they occur in large numbers They may migrate through the anus and invade the vagina. These worms are thread-like, measuring from 3 to 10 mm. in length, the female being the longer. The oxyuris vermicularis may propagate within their host, their ova requiring no intermediate host for their development.



duct, *ep*, excretory pore, *es*, esophagus, *g*, gubernaculum; *i*, midgut, ow_1 and ow_2, anterior and posterior oviducts, *r*, rectum; *sp*, buccal spears, *spi*, copulatory spicules, *t*, testis, *v*, vulva; *vs*, seminal vesicle (Faust's "Human Helminthology," Lea and Febiger)

Symptoms· The most constant and distressing symptom is itching of and around the anus (pruritus ani). There are also tenesmus, burning, restlessness and irritability. The itching is often worse during the night, thus disturbing sleep.

Ascaris Alata (*Ascaris Mystax*)· This is a species of roundworm found in the intestines of the dog and cat The worm is seldom found in man. The insect vectors are three different types of coffee flies (*simulii*).

Strongyloides Stercoralis: This is a minute common tropical worm, the female measuring about 2.2 mm. in length. It invades the duodenum and jejunum of man In massive infection they may invade the bile and the pancreatic ducts, the stomach, and the colon The eggs hatch out rhabditiform larvae which appear in the stool The outstanding symptoms are diarrhea and digestive disturbances. Occasionally there are no symptoms and the presence of infection may be discovered only by microscopic examination of the stool in which the parasites or their ova are found

Dracunculus Medinensis (Guinea or Medina Worm): This worm causes *dracontiasis* It is common in India, Persia, Africa and the East Indies. The adult female guinea worm measures from 15 to 80 cm by 0 5 to 1 7 m, while the length of the male is about 2 5 cm The developmental stages occur in an intermediate host, the fresh water copepods. e g, cyclops coronatus Man becomes infected by swallowing these crustaceans in drinking water It takes about one year before the adult stage is reached The adult worms reside in the connective tissue about the mesentery After copulation, the male worm dies and the gravid female migrates in search of water, invades the interstitial and sub-

cutaneous tissues, where it bores to the surface and discharges some secretion, forming a bleb which causes superficial ulceration in the center of which the head of the worm may protrude. On moistening the ulcer with water the parasites' uterus prolapses and a milky discharge containing many embryos is liberated from the base of the ulcer. The worm may often be palpated subcutaneously The ulcers appear most frequently in the lower extremities; they may also be found on the upper extremities, trunk, buttocks, scrotum, eyelids, tongue or other parts of the body. The adult worms usually appear during the summer months.

Symptoms of Dracontiasis: The acute symptoms are in the nature of an anaphylaxis, which occurs before ulceration takes place. There are fever, prostration, urticarial eruption, vasomotor collapse, diarrhea, dyspnea and a moderate eosinophilia. With the appearance of the worm subcutaneously, of the formation of a blister and of ulceration, the acute symptoms disappear. An intradermal test is said to have given a positive reaction in 85 per cent of cases.

Filaria: There are several species of filaria. They are threadlike minute worms and are carried by an intermediate host to man where they cause *Filariasis.*

Wuchereria Bancrofti or Filaria Bancrofti: Clinically this is the most important filaria. The adults of this species live in the lymphatics and in the region of the lymph nodes; they may also invade the testes, epididymis, spermatic cord, mammary gland and other parts of the body. The embryos invade the blood stream; they may be found in the lungs and thoracic blood vessels during the day, and in the peripheral blood stream during the night (nocturnal periodicity). The intermediate host is the *Culex fatigans* or other mosquitoes (SEE: p. 1090) which acquire the infection by biting an infected individual at night. After 10 to 40 days, the embryos have matured within the mosquito which may then transfer them to man where they develop into adult worms. The adult worm measures 30 to 100 mm by 0.2 mm, the female being the larger

Filariasis: In mild filarial infections there may be no symptoms When the filaria occur in large numbers and obstruct the lymphatics there may ensue lymphangitis with high fever, enlargement of the lymph glands, elephantiasis, chyluria and eosinophilia A definite diagnosis of filariasis can be made only when the larvae (microfilariae) are found in the blood, the urine or the chylous fluid. The disease is common in India, the West Indies, Puerto Rico, Southern China and the Pacific islands. In the Pacific, the insect vector is the *Aedes variegatus,* which bites during the day The filaria found there may be a different race or subspecies, since it is found in the peripheral blood stream during the day and does not exhibit periodicity (Low and Fairley).

Onchocerca Volvulus: The adults of this type of filaria may be found in the subcutaneous or connective tissue of man They occur in colonies chiefly in regions where lymphatic vessels converge, causing various lesions and tumors beneath the skin, and around the elbows, knees, ribs, iliac crests and great trochanters The tumors harbor the adult worms The microfilaria are also found in the tumors and in adjacent tissue These parasites are prevalent on the West Coast of Africa and are transmitted in the larval stage by the buffalo

gnat, *simulium damonsum* (SEE: p 1086).

Onchocerca Caecutiens: This filarial parasite is found in Guatemala and Mexico. It produces flat nodules upon the scalp and face associated with erysipelatoid swellings; it may also produce ocular disturbances and blindness. This filaria seldom affects other parts of the body.

swellings through a small incision. The insect vector is a fly belonging to the chrysops group. The parasites are found in West Africa.

Trichinella Spiralis: This is a small slender ovoviviparous worm. The male measures 1 4 to 1 6 mm. and the female 3 to 4 mm. in length. The embryo or muscle trichina is 0.1 to 1 mm. long and lies coiled up in a spiral form within

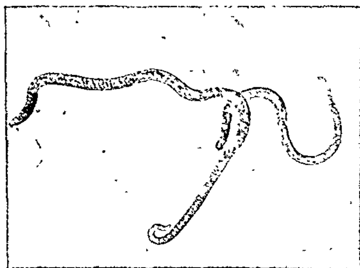


Fig 8—Trichinella spiralis Adult male (right) and female (left) developed in the duodenum ("Physician's Bulletin," Eli Lilly & Co)

Mansonella (*Demarquayi Ozzardi*) The adult worms live in the mesentery and the microfilariae in the circulating blood They occur in the West Indies and Northern South America.

Loa Loa (*Filaria Occuli, Filaria Loa*): This parasite lives underneath the conjunctiva and beneath the skin, particularly in the thoracic muscles. It causes fugitive subcutaneous swellings often the size of a hen's egg in various parts of the body (calabar swellings). These may last for a few days, then disappear and recur at another site The adult worm may be extracted from these

an ovoid capsule in the sarcolemma sheath of muscle fiber Man is infected with this parasite by eating infected uncooked or underdone pork products Smoking and salting do not destroy the larvae The larvae are also found in the muscles of pigs, rats, and bears Rats act as reservoir hosts Both pigs and rats acquire the parasite by eating infected human excreta, infected dead animals, and swill. When infected pork or bear meat is eaten by man the cyst wall surrounding the embryos are dissolved by the gastric juice, thus liberating them to mature and breed in the

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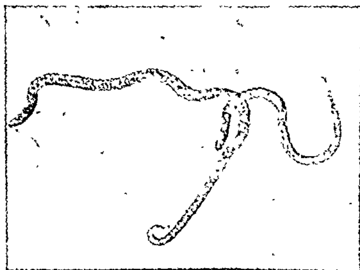


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buttocks This is followed by severe *hypochromic microcytic* anemia with marked eosinophilia. There is great physical and mental weakness, and, when the infection is acquired during *childhood, there is stunted somatic*, psychic and sexual development Mild cases *may show moderate* anemia, pale yellowish dry skin, some abdominal discom-

tropics. Both parasites are found chiefly in dogs and cats. Human infection is confined to the skin When these larvae penetrate the human skin, they do not enter the blood vessels, but burrow their way along the surface producing tortuous, linear or serpiginous lesions which cause intense itching. This condition is known as *Creeping Eruption, Creeping*

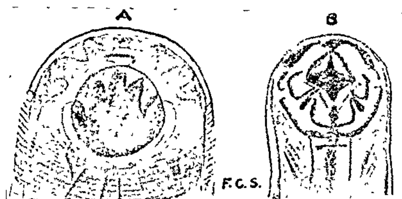


Fig 11—*A*, Dorsal view of Ankylostoma duodenalis *B*, Necator Americanus Both greatly magnified

fort, poor muscle power, dyspnea, and cardiac palpitation on mild exertion, causing a disinclination to work. Severe infection will show severe anemia, cardiac dilatation, weakness, stupor, generalized edema, ascites, and pleural effusion The skin is dry, the stomach is dilated and there may be vomiting and diarrhea The appetite is voracious A definite diagnosis is made when the ova are found in the stool The disease is endemic in India, Japan, the Pacific islands, Asia, South America, and various parts of the United States It is common where infected human feces is deposited on surface soil where people walk barefooted and also in mines where infected excreta disposal is unsanitary.

Ancylostoma caninum is found in both tropical and temperate climates and *Ancylostoma Braziliense* is found in the

Disease, Larva Migrans, or *Dermatitis linearis migrans*

Insects and Other Arthropods: Many of the arthropods that come in contact with humans may in themselves not be harmful, but are nevertheless dangerous because they may either be carriers of infectious microorganisms or be vectors in which parasites undergo one of their stages of development Among the arthropods known to be carriers or transmitters of disease-producing organisms are flies, mosquitos, mites, bugs, ticks, lice, fleas, and crustacea. Other arthropods may by their sting or venom cause local or general disease or death

Flies: Flies, irrespective of their specific type, may spread infection by disseminating infectious microorganisms.

The House Fly (Musca Domestica): This fly may carry typhoid fever, chol-

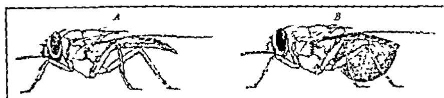


Fig 12—Glossina morsitans *A*, Before, and *B*, after feeding. Lateral view. (From Doflein after Austin) (MacNeal) (Stitt's "Diagnosis, Prevention and Treatment of Tropical Diseases" by Richard P. Strong. Copyright The Blakiston Company, Publishers)

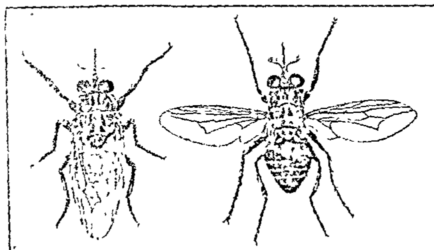


Fig 13—Glossina palpalis in natural resting position and with wings outstretched (MacNeal after Doflein) (Stitt's "Diagnosis, Prevention and Treatment of Tropical Diseases" by Richard P Strong Copyright The Blakiston Company, Publishers)



Fig 14—Chrysops discalis, showing the characteristic nonpigmented discal cell whence is derived its name (Stitt's "Diagnosis, Prevention and Treatment of Tropical Diseases" by Richard P Strong. Copyright The Blakiston Company, Publishers)

era, and bacillary dysentery organisms as well as the ova of tenia solium, ascaris lumbricoides and oxyuris vermicularis. It is also believed that the house fly may in part help to spread giardia, entameba hystolitica, leprosy and trachoma. It may also cause discomfort by depositing its eggs in wounds from which maggots develop and cause myiasis The favorable breeding place for this fly is human excrement, scraps of food, manure and filth of any kind that has some moisture The ova, wherever they are deposited, are hatched out in from one to five days into footless cream-colored maggots These larvae burrow into the ground, develop into pupa and emerge in from three to five days as adult flies.

The Lesser House Fly (Fannia Canicularis) usually breeds in human feces, old vegetables and vegetable refuse The live larvae are sometimes swallowed with the vegetables they infest and may cause intestinal myiasis.

The Tsetse Fly (Glossina) transmits Trypanosomiasis (Sleeping Sickness) (SEE: p 1069). There are about 20 species of Glossina; the most important are the *G. palpalis, G morsitans, G. tachinoides, G. brevipalpis,* and *G. swynertoni.* They are indigenous to Africa and Arabia. Some of the species live on the banks of rivers or lakes overhung with trees or bushes; others live in wooded country They are generally attracted by moving objects and will alight on pedestrians, running animals, automobiles, cyclists, etc.

The Stable Fly (Stomoxys Calcitrans) usually attacks animals and transmits systemic anthrax and malignant pustules. It may also transmit other pathogenic organisms by contact and is suspected of carrying the virus of poliomyelitis.

The Sand Flies or *Gnats* are of two different species, the *simulidae* and the *midges.* The *simulium damnosum* transmits the *onchocerca volvulus,* which is the filarial worm responsible for filariasis. An allied worm, *onchocerca coecutiens,* is said to be transmitted by the *Simulium avidum, S. mooseri,* and *S. ochraceum* These flies are prevalent during the spring and summer in many parts of the tropics and in Europe. The *Simulidae* are also known as buffalo gnats The female lays its eggs on waterweeds and stones in running streams

Among the *midges,* the most important is the *Phlebotomus* or *Pappataci sand fly.* It transmits phlebotomus fever, Oriental sore, and probably Kala Azar. Oroya fever is spread by *Phlebotomus noyuchii, a very small hairy fly that moves* about in short flights much like a flea

The midge fly breeds in dark damp places, such as cellars, caves, dugouts, under damp stones, damp stone walls and in cracks and fissures in damp soil The eggs hatch into minute caterpillar-like larvae which live in organic matter

The Deer Fly (Chrysops discalis) is suspected as being one of the transmitters of tularemia. The other and more common vectors are ticks.

Carcase (Carcass) Flies include the Blow fly, the Blue Bottle fly, the Green Bottle fly and the Gray-colored hairy fly and the American Screw fly They are usually found in decomposing *flesh and* other decomposing matter where they *deposit their ova*; these may hatch in the intestines and cause intestinal myiasis Some of these flies may also deposit their ova on wounds and upon any pus-discharging surface. Their maggots, if not infected by pyogenic *organisms, are* at times beneficial in cleaning up certain wounds and stimulating healing. How-

ever, some maggots may enter the external auditory canal in cases of otorrhea, or the nares in cases of ozena, and find their way into the brain or sinuses and cause meningitis. These flies as well as other winged pests may spread disease by disseminating infectious organisms from various sources, thus acting as mechanical carriers.

The Tumbu Fly (Cordilobia anthropophaga) deposits its ova upon the clothing and skin of the unwashed The ova hatch out as maggots (*Ver du Cayor* or *African skin maggots*) and penetrate the skin, causing subcutaneous boil-like lesions.

The Congo Floor Maggot Fly (Auchmeromyia luteola) is a fly resembling the tumbu fly Its maggots are known as the *Congo Floor Maggots,* they are the only dipterous larvae known to suck human blood These maggots are found in large numbers on the ground floors of huts where people sleep on the ground. They are prevalent in the Belgian Congo and in tropical and subtropical East Africa. No definite disease is identified with this fly or its larvae, but the blood-sucking proclivity of the maggots may cause severe anemia

Myiasis: This is a disease caused by the presence of fly maggots in some parts of the body. *Cutaneous myiasis* is usually caused by larvae that invade wounds, or sores, very few pierce the skin. They may be found upon the surface of the infected skin, the nasal folds, ears, eyes, corners of the mouth, and the genital orifices and may occasionally gain entrance into the body through these orifices. *Intestinal myiasis* may be caused by accidentally swallowing ova or maggots with food or drink, or by direct infection by ova which can only develop in living tissue. The common cau this type of myiasis is the larvae o Tumbu fly (*Cordylobia anthropopha* The diagnosis of cutaneous myiasi self-evident Intestinal myiasis may c severe diarrhea, dysentery, general w ness and emaciation.

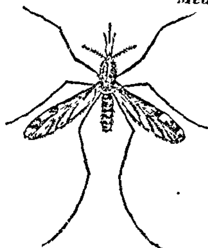
Mosquitoes: The two import groups of mosquitoes are the *Anophel* which are responsible for the varic types of malaria, and the *Culicini*, whi are responsible for the transmission yellow fever, dengue and the filarias due to Wuchereria bancrofti; each these main groups has numerous specie which are indigenous to many parts o the world and transmit various diseases The females only of these species suck blood and therefore are the carriers of the infection.

Differential Points Between Male and Female and Between the Two Groups The females of both the Anophelini

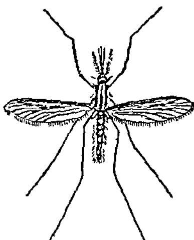


Fig 15—Resting posture of mosquitoes 1 and 2, Anopheles, 3, Culex pipiens (After Sambon) From P. H Reports (Stitt's "Diagnosis, Prevention and Treatment of Tropical Diseases" by Richard P. Strong Copyright The Blakiston Company, Publishers)

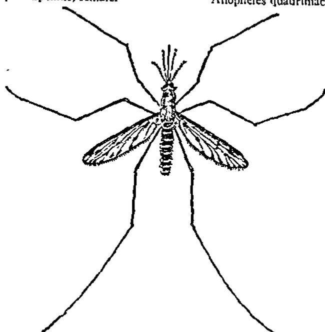
and Culicini have sparsely haired antennae, while the male antennae are densely haired and plumelike. The female Anophelini have polypi as long, or nearly as long, as the proboscis, while the probosci of the Culicini are very much shorter The resting positions of the two types also differ. The Anophelini usually stand with their heads down



Anopheles punctipennis, female.



Anopheles quadrimaculatus, female



Anopheles crucians, female

[illegible]
old the end spot is usually missing

and their bodies pointing upwards at an angle of 45 degrees, while the Culicini rest nearly parallel to the surface, their rear end and head being somewhat depressed The Anopheline mosquitoes are very much less scaly than the other group.

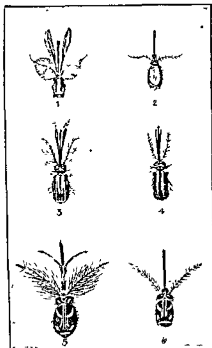


Fig 17—Heads of mosquitoes 1 and 2, male and female Culex quinquefasciatus, 3 and 4, male and female Anopheles, 5 and 6, male and female Aedes aegypti (After Stitt) From P H Reports (Stitt's "Diagnosis, Prevention and Treatment of Tropical Diseases" by Richard P Strong *Copyright The Blakiston Company, Publishers*)

All types of mosquitoes lay their eggs in quiet water After a few days the eggs hatch into the so-called wrigglers in the water which undergo further stages of development to emerge finally in several weeks as adult mosquitoes (depending upon the temperature of the water and the food supply)

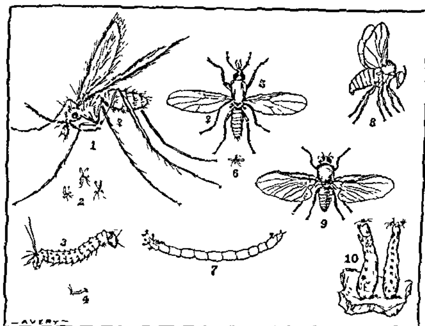
The Anophelini Mosquitoes (Malaria carriers) There are about 50 or more species of the Anopheline group; some are constant carriers wherever found, others are carriers only in some localities and not in others, while a third variety, although susceptible to infection, is apparently of little epidemiological importance The variability of their infectiveness probably depends upon variability of their habits and habitat The four species of malarial parasites, namely the *Plasmodium vivax* and *P. ovale* responsible for benign tertian malaria, the *P malaria* causing quartan type malaria, and the *P. falciparum* which produces a malignant, subtertian or estivo-autumnal fever are transmitted by infected Anopheline mosquitoes In order to become infective the mosquito must first bite a person that has both male and female malarial parasites in the circulating blood These fertilize in the mosquito's stomach, and the fertilized forms find their way between the stomach cells, form cysts on its outer wall and mature in about eight days The cysts rupture in the body cavity of the mosquito, liberating the sporazoids, these travel to the salivary glands and are injected through the proboscis into the blood stream of the bitten person in whom, ten days later, the parasites are found in the erythrocytes, and malaria becomes manifest

The Culicini Mosquitoes: There are 20 or more species of mosquitoes belonging to the Culicini group The *Aedes aegypti* (*Stegomyia fasciata*) is the common transmitter of the filtrable virus causing yellow fever in man In order to transmit yellow fever the mosquito must bite a yellow fever sufferer during the first three days of his illness. Then, after nine to twelve days and until its death, the mosquito is capable of transmitting the disease by its bite. In Africa

and in locations where Jungle Yellow Fever is prevalent and where the *Aedes aegypti* does not exist, the yellow fever virus is transmitted by other species of the Aedes type. Those who have recovered from yellow fever, even in mild form, possess a lifelong immunity to the disease.

cus brevipalpis, transmits "Rift Valley Fever," a fatal epizootic disease occurring in certain parts of East Africa (Kenya) and affecting ewes and lambs It may be transmitted to man in whom it is not fatal.

The Eastern and Western strains of encephalomyelitis virus may be trans-



(Stitt's "Diagnosis, Prevention and Treatment of Tropical Diseases" by Richard P. Strong Copyright The Blakiston Company, Publishers.)

Aedes aegypti and probably also *Aedes albopictus* and *Armigeres obturbans* are the transmitters of the filtrable virus responsible for Dengue. An infected mosquito is capable of transmitting the disease throughout its life. *Aedes aegypti* and *Culex fatigans* and occasionally several others of the Culicini and Anopheleni are transmitters of *Wuchereria bancrofti* causing filariasis and various symptoms of lymphatic obstruction Another Culicini mosquito, the *Taeniorhyn-*

mitted experimentally by various species of the Aedes mosquito.

Mites or Chiggers and Gnats: These usually produce only temporary skin irritation Occasionally, particularly the Japanese mite may become infected with Rickettsialike organisms while feeding on field mice and transmit "Flood fever," or "Tsutsugamushi" A small gnat, the *Hippelates pallipes,* is suspected of being the mechanical vector of the yaws.

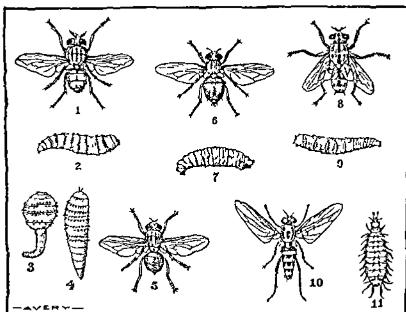
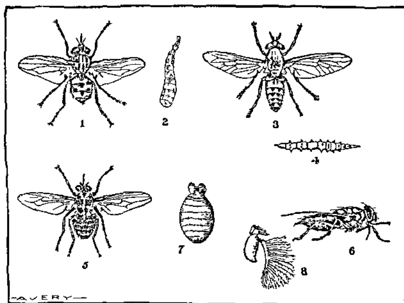


Fig 19—Insects in which the larvae stage is important. *1*, Chrysomyia macellaria, *2*, C. larva; *3*, Dermatobia hominis larva, early stage (ver macaque); *4*, D. hominis larva, later
', A luteola larva; *8*,
11, A pluvialis larva
by Richard P. Strong



F

Bedbugs (*Cimex lectularius* and *C. hemiptera*): Among the bugs that attack man, the bedbug is the commonest There are several species that are suspected of transmitting disease. *Cimex hemipterus rotondatus*, an Indian species, is suspected of harboring Leishmania donovani, causing Kala Azar. The Triatoma group belonging to the reduviid bug family transmits trypanosomiasis and probably Chagas' disease

Bedbugs are also suspected of carrying plague, anthrax, relapsing fever and typhoid fever. They are employed as experimental hosts for *Trypanosoma cruzi* and *Leptospira icterohemorrhagiae*.

Both males and females suck blood; they are nocturnal pests. During the day they usually hide in cracks in the walls, floor, furniture, beds, and bedding, or in any sheltered place Bedbugs may travel long distances, from house to house, or tent to tent, and may remain for nine months or longer without food. *They seem to be more numerous in cold* than in hot climates. The adult bug may survive freezing temperature for some time.

Ticks (*Arachnids*): These are of two general types, hard ticks (*Exodidae*) and soft ticks (*Argasidae* or *Argantidae*) Some ticks require three intermediate hosts for their development, some, two, and others, one intermediate host. They are also classified according to their structures and habitat. Ticks may be carriers of Rickettsia, Spirillae, bacteria and other pathogenic organisms

They usually infest the skin of dogs, rabbits, and other furry animals from whom some may fall off and become adherent to stalks of grass, plants or weeds.

The **Dermacentor Andersoni** (*D. Venustus*) is a hard tick and is a carrier of the Rickettsia responsible for Rocky Mountain Spotted Fever (Dermacentroxinus rickettsii). The dog tick (*Rhipicephalus sanguineus*) may spread Fievre Boutonneuse (Marseilles Fever), a form of acute ascending paralysis especially of children Another hard tick (*Ixodes ricinus*) is responsible for Louping Ill, a form of encephalomyelitis of sheep Human infection may occur in contacts.

The *Dermacentor Andersoni* and the *Dermacentor variabilis* have also been found to carry the bacterium tularense.

The *Ornithodorus moubata* transmit the spirillum responsible for relapsing fever. They are blind ticks whose feeding habits resemble those of the bedbug They are indigenous to Africa and are also found in Central Asia, India, Arabia, Persia, Southern Spain, and in the tropical regions of the Americas These ticks live in native huts and rest houses During the day, they hide in crannies of walls, floors, roofs and other dark places, *and at night they migrate in quest of* food, which is human or animal blood

Lice (*Pediculi*). Lice affecting man are of three types· *Pediculus capitis* (head louse), *pediculus corporis* (body louse), and *phthirius pubis* (the crab louse) Lice are responsible for several serious epidemic diseases They transmit *typhus fever, trench fever* and *relapsing fever*, and cause local skin irritations It is of great importance to prevent the occurrence of lice or to exterminate them in camps, institutions, and in places where numbers of people live in close proximity.

Fleas (*Siphonaptera*): There are various species of fleas each having a predileotion for a definite host. The rat flea (*xenopsylla cheopis*) transmits *Bubonic plague* and *Brill's disease* (endemic typhus). The human flea (*Pulex*

irritans) is the only flea of which man is the usual host, though any type of flea may occasionally affect man Of the various types of fleas that affect rats, mice, dogs, cats, squirrels, etc, the *xenopsylla cheopis* is the most important from the standpoint of infection Bacot[1] showed that the larval stage may last from 12 to 84 days, and the cocoon stage from 7 to 182 days

Fleas' eggs, after being laid, fall to the ground; they are usually found in sleeping places of animals The eggs hatch into footless sparsely covered hairy larvae which live in the dust of floors and feed on organic matter After about two weeks the larva spins a cocoon in which it pupates, and after another two weeks it emerges as an adult flea The length of the various stages of development depends chiefly upon the temperature, being faster during the summer and slower in the winter.

Other winged or wingless nonvenomous insects are not identified with the transmission of any specific disease, though bees, hornets, moths, butterflies, dragonflies, spiders, ants, roaches, and itch mites may act as mechanical vectors, that is, spreading disease to man and animal by infecting food or drink with pathogenic organisms that may adhere to their bodies Venomous arthropods, such as various types of spiders, scorpions, tarantulas, certain caterpillars, wasps, bees and certain ants may by their sting cause painful local lesions, systemic infection and at times death.

The Crustacea: The cyclops coronatus, copepods and various species of crabs and crawfish serve as secondary intermediate hosts of certain intestinal nematodes, cestodes and flukes. Oysters and clams may harbor the typhoid bacilli and transmit typhoid fever

Fungi and Monilia—Mycotic and Monilia Infections

Fungi and monilia may cause systemic disease when they affect internal structures, or they may produce various skin affections when they remain upon the surface Diseases caused by fungi are classified as the mycoses or mycotic infections and those caused by monilia as moniliasis or monilia infection

The Mycoses

Actinomycosis (Ray fungus disease, Lumpy Jaw). This is an infection caused by a ray fungus, *streptothrix actinomyces* or *actinomyces bovis* The disease is more common in cattle and is transmitted to man by cattle or their pelt It starts as a local infection which later may become generalized causing granulomatous lesions These are characterized by the formation of multiple small abscesses which communicate and form discharging sinuses, or there may be large abscesses with induration and granulation areas The symptoms depend upon the areas affected The jaw and the adjacent structures are the more common sites, other structures that may become involved are the abdomen and its viscera, the lungs and pleura, the brain, or the skin.

Aural actinomycosis is characterized by toothache, dysphagia, and partial trismus Later there develop swelling and induration of the tongue (macroglossia) at the angle of the jaw of the thyroid and of adjacent structures, which suppurate and discharge pus containing yellow masses.

[1] *Cited by* W P MacArthur · *Medical Diseases in Tropical and Subtropical Areas*, 1942

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Histoplasmosis of Darling: This is a severe, often fatal, disease caused by a fungus, the *Histoplasma capsulatum,* which generally invades the reticulo-endothelial cells, and may also be found in the blood and other tissues. The fungus may appear in two forms, one, yeastlike when recovered from the blood or reticuloendothelial tissue, the other, a mycelial form when cultured outside the body.

Symptoms: The outstanding manifestations are continued fever, splenomegaly, anemia with leukopenia. It may affect the lungs causing widespread lesions resembling metastatic malignancy Recently[1] several cases of histoplasmosis were reported in adults and in children. The diagnosis may be made by finding the organisms in stained smears or sections, or by cultures The disease often occurs in conjunction with some chronic affection such as diabetes, cancer or other chronic diseases. It may, however, occur in apparently otherwise normal persons Cases were reported from temperate as well as tropical regions.

Coccidioidal Granuloma (California disease, coccidioides) This disease is caused by a hypomycetic fungus, the *coccidioides immitis;* it may run an acute, subacute, or chronic course, and resembles blastomycosis. It may affect the skin, causing nodular lesions, abscess and gummatous ulcers containing thick pus It may also affect the lungs, causing lesions resembling tuberculosis, and occasionally the meninges and the bony structures may develop suppurative lesions The discovery of the coccidioidiae in the lesions or in the pus or a positive coccidioidin intradermal test is diagnostic.

[1] Wright-Hachtel Am Int Med. 15: 309, 1941 Meleney, H E Am Rev Tuberc 44. 240, 1941 Rhodes, Conant and Glesne: Jr. Pediat. 18 235, 1941.

Cryptococcosis and Torulosis: These are produced by a yeastlike organism termed *saccharomycosis.* The torula infection, according to Low and Fairley, is caused by the *Torula histolytica.* Benham suggests the term *cryptococcus hominis* for organisms of this type. The lesions have a predilection for the central nervous system, but may also affect the subcutaneous tissue, bone and viscera. It is characterized by the formation of gelatinous cystlike lesions; these contain the organisms. In cerebrospinal involvement the organisms resembling lymphocytes are found in the cerebrospinal fluid. Cases of Torula meningoencephalitis were reported[1] in which the yeastlike organisms were recovered from the spinal fluid.

Rhinosporidiosis: This is a chronic disease characterized by the formation of nasal polypi and papillomatous lesions upon the conjunctivae, lacrymal sacs and cheeks The causative organism is a vegetable mold belonging to the order of phytomycetes, the *rhinospordium seeberi.*

The Moniliases

The monilia fungi are, for the most part, saprophytic, and are widely distributed in nature. To the genus monilia belongs a large number of different species which, because of the similarity of their behavior, may for clinical purposes be grouped as a single species. On glucose-containing media they grow upon the surface, eventually forming large creamy plaques with raised edges. The diagnosis of moniliasis is either based

[1] Stiles, W. W.: Jour. A. M. A. 115: 601, 1940. Curtis, A. N.: Jour. A. M. A. 116: 1633, 1941.

In *abdominal actinomycosis* the more common site is the cecum and appendix, causing appendicitis. The infection may spread to the liver, causing enlargement and abscess formation; it may also affect other abdominal viscera and the peritoneum. When the abdominal wall becomes involved, suppurative sinuses may result.

Pulmonary actinomycosis causes lesions in the lungs resembling atypical pneumonia, tuberculosis or malignancy. The symptoms usually start with pleural pain; later there develop cough with fetid expectoration and, at times, hemoptysis.

Cerebral actinomycosis causes symptoms of space-taking lesions and meningeal irritation.

In *skin actinomycosis,* granulomatous lesions occur on the affected site. This may occur in conjunction with lesions in other sites.

The disease usually runs a moderately protracted course.

The diagnosis depends upon the discovery of the "sulfur granules" containing the mycelia in the pus, sputum or in other secretions. The fungi may reside in the normal mouth, in tonsillar crypts, or in carious teeth without causing pathologic lesions.

Mycetoma (Madura foot, Pseudoactinomycosis): This is a chronic granulomatous infection especially of the feet, it rarely affects the hands or other parts of the body. There appear marked swelling and multiple abscesses which connect with deeper sinuses discharging a foul-smelling, oily pus containing various-colored fungoid granules. The disease is common among the natives in the rural districts of Northern Africa, China, the West Indies, South America, and is occasionally seen in the United States

Sporotrichosis: This is a chronic infection by the *sporotrichum schenki* and *S. beurmanni,* affecting the skin and the underlying tissue, usually of the hand or foot and causing gummalike nodules, abscesses and ulcers. The disease spreads by way of the lymphatics After forming subcutaneous cold abscesses along the infected lymph channels, there may develop indolent fungating ulcers. The lesions are painless and infectious and may be transmitted by infected persons or animals.

Blastomycosis (Gilchrists' disease, Chicago disease). This is a chronic granulomatous and suppurative process affecting the skin, the subcutaneous tissue, the lungs or other internal viscera. It is caused by some species of yeastlike blastomycoids. The skin lesions may be papuloulcerative or nodular; there may occur tumorlike granulomata discharging pus, the lesions may resemble tuberculosis or syphilis. The pulmonary lesions cause cough and expectoration of bloody mucopurulent material. Other symptoms depend upon the affected area. The diagnosis is made by recovering the blastomycetes from the pus or from the lesions. The disease was found in fairly large numbers in Chicago, Ill It is now also found in other portions of the United States, in Canada, and in Puerto Rico

Streptothricosis: This is a fungus infection by the *streptothrix asteroides,* the lesions resembling those of actinomycosis They are suppurative, forming abscesses and granulation tissue. The lungs are the usual site of the infection where it may cause bronchopneumonia, abscess, gangrene and empyema. The diagnosis is made by the discovery of the streptothrix in the sputum or pus.

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upon or confirmed by the finding of monilia in the lesions or in the secretions or by culture.

The monilia group commonly affect the skin; they may also affect the mucous membrane and the viscera.

Cutaneous Moniliasis: The growth of monilia is stimulated by warmth and moisture; therefore infection is commonest in the folds of the skin such as underneath the breasts, in the axillae, in the crotch and in the perineal folds It may also be found around the rectum and the vagina. The lesions consist of patchy, slightly raised areas of erythema often made up of vesicles and pustules The edges are slightly raised and the patches have a tendency to become confluent. There is usually burning or intense itching. Perspiring feet and hands may develop vesicular noninflammatory lesions resembling epidermophytosis Affection of the fingernails may cause paronychial swellings; the nails become lusterless, friable, thickened, ridged and discolored.

Thrush (parasitis stomatitis): Affection of the mucous membrane is best exemplified by thrush It occurs upon the mucous membranes of the mouth The mouth is dry and there are scattered small white patches resembling milk curds distributed over the gums, tongue, cheeks and lips This is associated with considerable burning

Pulmonary Moniliasis: In this the bronchi are chiefly affected, though the infection may spread to the vesicular structures The symptoms are those of bronchitis or of bronchopneumonia The physical signs may reveal unilateral or bilateral involvement Culture of the sputum may reveal the cause of the infection